GASTROENTEROLOGY

ABSTRACTS AND CITATIONS

NATIONAL INSTITUTE OF ARTHRITIS, METABOLISM, AND DIGESTIVE DISEASES

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PREFACE

GASTROENTEROLOGY ABSTRACTS and CITATIONS is a publication of the National Institute of Arthritis, Metabolism and Digestive Diseases. This specialized information medium has been initiated to fill an existing great need in the field of gastroenterology and to assist the Institute in meeting its obligations to foster and support laboratory and clinical research into the nature, causes, and therapy of diseases of the gastrointestinal tract. Publication GASTROENTEROLOGY ABSTRACTS and CITATIONS makes available citations of all current papers relevant to this field from medical journals published throughout the world. Approximately one-third of the citations dealing with the major aspects of gastroenterology have accompanying abstracts.

The issuing of GASTROENTEROLOGY ABSTRACTS and CITATIONS under the auspices of the National Institute of Arthritis, Metabolism, and Digestive Diseases will provide a much needed current awareness tool to scientists and will facilitate greater integration of research and clinical efforts in this field. The number and great diversity of publications in the area of gastroenterology makes it imperative that an appropriate service be available to investigators and practitioners so that they may be apprised of progress with a minimum of delay. Our aim is to provide the readers with a readily systematized compilation of current published work. The publication will provide the greatest usefulness if these interested investigators will contribute their ideas and comments for consideration whenever possible.

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NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations (with some modifications) found in *International Standard ISO833, 1974*, are used. The two-letter state abbreviations are based on the United States Postal Service Code.

LANGUAGE ABBREVIATIONS

Afrikaans	Afr.	Georgian	Geo.	Persian	Per.
Albanian	Alb.	German	Ger.	Polish	Pol.
Arabic	Ara.	Greek	Gre.	Portuguese	Por.
Armenian	Arm.	Hausa	Hau.	Pushto	Pus.
Azerbaijani	Aze.	Hebrew	Heb.	Rumanian	Rum.
Belorussian	Bel.	Hindi	Hin.	Russian	Rus.
Bengali	Ben.	Hungarian	Hun.	Serbian	Ser.
Bulgarian	Bul.	Icelandic	Ice.	Slovak	Slo.
Burmese	Bur.	Indonesian	Ind.	Slovene	Slv.
Chinese	Chi.	Interlingua	Int.	Spanish	Spa.
Croatian	Cro.	Italian	Ita.	Swahili	Swa.
Czechoslovakian	Cze.	Japanese	Jpn.	Swedish	Swe.
Danish	Dan.	Kirghiz	Kir.	Tagalog	Tag.
Dutch	Dut.	Korean	Kor.	Tamil	Tam.
English	Eng.	Latin	Lat.	Telugu	Tel.
Esperanto	Esp.	Latvian	Lav.	Thai	Tha.
Estonian	Est.	Lithuanian	Lit.	Turkish	Tur.
Finnish	Fin.	Marathi	Mar.	Ukranian	Ukr.
Flemish	Fle.	Masai	Mas.	Urdu	Urd.
French	Fre.	Multilingual	Mlt.	Vietnamese	Vie.
Gaelic	Gae.	Norwegian	Nor.	Welsh	Wel.

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	ml, µl	milli-, microliter(s)
ADP	adenosine diphosphate	mm	millimeter(s)
AMP	adenosine monophosphate	mOsm	milliosmolar; milliosmeter(s)
amp	ampere	msec	millisecond(s)
ATP	adenosine triphosphate	mV	millivolt(s)
A	angstrom	min	minute(s)
BSP	sulfobromophthalein	MTD	maximum tolerated dose
C	degrees centigrade	NAD	nicotinamide adenine dinucleotide
cal	calories	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter		
cm	centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
dl	deciliter		
DNA	deoxyribonucleic acid	NADPH	reduced nicotinamide adenine dinucleotidephosphate
ED_{50}	median effective dose(s)		
EDTA	ethylenediamine tetraacetic acid	ng	nanogram (10^{-9})
e.g.	for example	nm	nanometer(s)
EEG	electroencephalogram	Pa	ambient pressure
ESR	erythrocyte sedimentation rate	PAS	periodic acid - Schiff
g	gram(s)	pg	picogram (10^{-12})
µg	microgram(s)	pgEq	picogram equivalent(s)
Hz	Hertz	p.o.	orally
hr	hour(s)	ppb	parts per billion
Ig	immunoglobulin(s)	ppm	parts per million
i.m.	intramuscular	r	Roentgen
i.p.	intraperitoneal	RBC	red blood cells (erythrocytes), red blood count
IU	international unit(s)		
i.v.	intravenous	resp.	respectively
kcal	kilocalorie(s)	RNA	ribonucleic acid
kg	kilogram(s)	s.c.	subcutaneous
K_m	Michaelis constant	sec	second(s)
LD_{50}	median lethal dose(s)	SGOT	serum glutamic-oxalacetic transaminase
LDH	lactic acid dehydrogenase		
m	meter(s)	SGPT	serum glutamic-pyruvic transaminase
M	molar; mole(s)		
mM	millimolar; millimole(s)	U	unit(s)
µM	micromolar; micromole(s)	UV	ultraviolet
ma	milliampere(s)	V	volt
mCi, µCi	milli-, microcurie(s)	WBC	white blood cells (leukocytes), white blood count
mEq	milliequivalent(s)		
mg	milligram(s)	yr	year(s)

PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

0778 CYTOCHEMICAL STUDY OF INTESTINAL ENDOCRINE CELLS IN RABBIT FETUSES DURING IN VITRO INDUCED DEGENERATION AND DIFFERENTIATION. (Fre.) Lefranc, G.; Pradal, G.; L'Hermite, A. L.; Andre, M. J.; Ducluzeau, A. (Laboratoire d'histologie et Embryologie, Faculte de Medecine, F-44 Nantes, France). *Histochemistry* 53(2):157-163; 1977.

In vitro culture of fragments of the intestinal mucosa of rabbit fetuses in solid media induces epithelial degeneration followed by regeneration. At the end of 3 days, degenerated necrotic cells sloughed off in the intestinal lumen and regenerating cells in contact with the chorion could be observed on the same histological section. The histochemical characteristics of the serotonin cells were studied and related to the stages of differentiation. The cells acquired successively the following characteristics: fluorescence induced by formaldehyde (FIF), then argyrophilia, then affinity for silver salts. *In vivo* studies have shown that FIF and argyrophilia appear simultaneously. These *in vitro* studies suggest an extra step in the differentiation of the serotonin-producing cells of the intestine that may exist *in vivo* but has not as yet been observed.

0779 ENDOCRINE CELLS IN THE HUMAN FETAL SMALL INTESTINE. (Eng.) Moxey, P. C.; Trier, J. S. (Peter Bent Brigham Hosp., 721 Huntington Ave., Boston, MA 02115). *Cell Tissue Res.* 183(1): 33-50; 1977.

The time of appearance and ultrastructural features of enteroendocrine cells (EECs) were studied in the human fetal small intestine between 9 and 22 weeks of gestation. Thirteen distinctive EECs were identified in fetal small intestine. Two of these, not found in normal small gut, appeared within the stratified epithelium of the proximal small intestine at 9-10 weeks. They were arbitrarily termed "primitive" and "precursor" cells. As in all fetal EECs, the pale cytoplasm of the "primitive" cell contained a distinctive population of secretory granules (SGs). Primitive cell SGs averaged 200-330 nm; some had dense cores with lucent halos, while others were filled with a homogeneous dense or flocculent material. The SGs of the "precursor" cells were larger, averaging up to 1 μm in diameter, and their contents varied in electron density. A third group of cells not described in adult small bowel was arbitrarily termed "transitional" cells. These had two populations of SGs; one resembled the SGs of the "precursor" cells, and the other resembled the SGs of some of the specific adult type EECs. Transitional enterochromaffin (EC), S, I, and G cells were seen. In addition, mature appearing EC, S, G, I, L, D, and D_1 cells were identified by 12 weeks of gestation. The "primitive," "precursor," and "transitional" cells may represent sequential developmental precursors of adult type EECs.

0780 RABBIT ILEAL MUCOSA EXPOSED TO FATTY ACIDS, BILE ACIDS, AND OTHER SECRETAGOGUES: SCANNING ELECTRON MICROSCOPIC APPEARANCES. (Eng.) Gaginella, T. S.; Lewis, J. C.; Phillips, S. F. (Coll. Pharmacy, Ohio State Univ., Columbus, OH 43210). *Am. J. Dig. Dis.* 22(9):781-790; 1977.

Segments of rabbit ileum were examined by scanning electron microscopy (SEM) after exposure to various compounds known to stimulate small intestinal fluid secretion. After perfusion with 10 mM ricinoleate, striking changes were observed at villus tips and on the apicolateral surfaces of villi; erosions at tips were confirmed by light microscopy of the same pieces of tissue examined by SEM. The apicolateral surfaces of the villi had a "cracked clay" appearance. The appearances after ricinoleate were reversed in part during perfusion with control buffer for 2 hr. Statistical comparisons between control perfused, treated, and reversal tissues showed that the "damage" produced by ricinoleate, and its reversal, was statistically significant. Tissues treated with other intestinal secretagogues, oleic acid (10 mM), deoxycholic acid (4 mM), cholic acid (4 mM), dioctyl sodium sulfosuccinate (6 mM), and cholera toxin (1 μg/ml) also showed surface changes by SEM. Sodium oleate and sodium deoxycholate produced villus tip changes, dioctyl sodium sulfosuccinate and sodium cholate resulted in the "cracked clay" pattern, and cholera toxin produced both changes. Fluid secretion, produced by mannitol was not associated with "damage." Structural changes of the mucosal surface after exposure to intestinal secretagogues should be considered as one possible mechanism of impaired fluid transport.

0781 COLONIC MUCOSAL ATROPHY INDUCED BY A LIQUID ELEMENTAL DIET IN RATS. (Eng.) Janne, P.; Carpentier, Y.; Willems, G. (Hopital Saint-Pierre, rue Haute 322, B. 1000 Brussels, Belgium). *Am. J. Dig. Dis.* 22(9):808-812; 1977.

The effect of feeding a liquid elemental diet (Vivonex®) for 4 weeks on the weight, the total crypt cell population, and the proliferation parameters of the colon was studied in male Wistar rats. Both mitotic activity and DNA synthesis activity were decreased ($p<0.01$) in the colonic mucosa during the administration of the diet, in comparison with that in controls. The weight of the colon was decreased ($p<0.01$) from the first week of treatment. After 4 weeks, a 75% decrease in the cell population of mucosal glands was observed, indicating that there had been considerable atrophy of the colonic mucosa. This atrophy was probably mediated by a reduction in the proliferative activity of the stem cells in the mucosal glands.

0782 MORPHOLOGY OF THE ISOLATED OXYNTIC CELL AND SOME PHYSIOLOGICAL PARAMETERS. (Eng.) Ito, S.; Munro, D. R.; Schofield, G. C. (Harvard Medical Sch., Boston, MA 02115). *Gastroenterology* 73(4):887-898; 1977.

The morphology of the isolated mouse oxyntic cell is described and compared with that of the intact stomach. Isolated oxyntic cells adopt a variety of morphological configurations. Some cells retain their polarity, so that a portion of the surface membrane is formed by laterobasal membrane and the remainder by luminal surface membrane with secretory or intracellular canaliculi opening to the outer surface. Other cells present a smooth outer surface and a completely internalized canaliculus. There are also oxyntic cells in which the whole surface membrane is covered with numerous microvilli resembling those of the luminal surface of oxyntic cells *in situ*. In cells where both laterobasal and luminal membrane can be distinguished, a junctional region that shows an intermixing of these two types of surface membranes is seen. Intracellular canaliculi of isolated oxyntic cells may be either distended and lined by microvilli or virtually collapsed and occluded by numerous microvilli. Evidence of functional activity by isolated oxyntic cells includes a small but consistent movement of chloride toward the cathode when a polarizing potential is applied to isolated cells forming a monolayer. Uptake studies with ^{3}H-leucine indicate that isolated oxyntic cells avidly incorporate this substrate and apparently metabolize it. Current studies on respiration by isolated oxyntic cells reveal a doubling of oxygen consumption when succinate is added to the incubating medium. However, oxygen utilization by isolated cells from acetazolamide-inhibited mice is only one-half that of nontreated control cells.

0783 FROG GASTRIC TUBULAR CELLS: ISOLATION, CULTURE, AND SOME PROPERTIES. (Eng.) Kasbekar, D. K.; Blumenthal, G. H. (Georgetown Univ. Sch. Medicine and Dentistry, Washington, DC 20007). *Gastroenterology* 73(4):881-886; 1977.

The methods used for obtaining primary cultures of the gastric tubular cells from *Rana catesbeiana*, the maintenance of their differentiated state under culture conditions of several weeks, and some of their properties are described. The problem of microbial contamination in the amphibian cells was overcome by incubating the cells in medium containing an antibiotic mixture. During the first few days after isolation and culture, cell surface damage was manifested by enhanced leakage of intracellular pepsinogen and a lack of response to known gastric secretagogues with respect to pepsinogen release and respiration. After several days in culture, the cellular pepsinogen levels increased slightly but significantly, and the spontaneous loss of pepsinogen from the cells showed a marked decline, indicating repair of the initial membrane or surface damage. The intact cells possessed *p*-nitrophenylphosphatase activity. After several days in culture, the respiratory activity became sensitive to the serum concentrations of the culture medium and was relatively stable. Antibiotics used in establishing primary cultures reversibly inhibited respiration and pepsinogen synthesis. A variable response to the gastric secretagogues was observed with regard to respiratory and pepsinogen secretory activities, whereas the *p*-nitrophenylphosphatase activity remained unaffected. There was no indication that the cells divided under the present culture conditions. Although these cells did not attach firmly to the surface of plastic or glass culture flasks, preliminary evidence indicates that collagen gel may provide an appropriate matrix for cell attachment. Further studies with a reconstituted epithelium comprised of a homogeneous gastric tubular cell population are indicated.

0784 TERMINATION AND LUMEN OF THE HEPATOPANCREATIC EXCRETORY DUCTS. A MORPHOMETRIC STUDY. (Ger.) Muller, M.; Putzke, H. P. (Institut fur Allgemeine und Spezielle Pathologie, Wilhelm-Pieck-Universitat, Strempelstrasse 14, DDR-25 Rostock, E. Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 37(1):1-6; 1977.

The termination and lumen of the hepatopancreatic excretory system was studied in 200 autopsy cases. Common papilla was found in 183 cases and common pancreaticobiliary duct in 141. The hepatopancreatic excretory system had type y termination in 70.5% of all cases, type v in 21%, and type u in 8.5%. The average length of the ampulla of Vater was 5.4 mm. The diameter of the pancreatic duct was 7.3 mm in the head of the pancreas, 5.7 mm in the body, and 2.5 mm in the tail. The diameter of the choledochal duct was 3.5 mm at its entry into the ampulla of Vater and into the duodenum, 12 mm at 2-cm distance proximally, and 16.5 mm at 6-cm distance proximally. No correlation was found between the lumen of the pancreatic duct and the type of the termination, but the size of the lumen increased with age.

0785 ULTRASTRUCTURAL STUDY OF COLONIC MUCOSA INVASION BY E. histolytica. (Spa.) Trevino, N.; Cruz de Lavin, E.; Tanimoto-Weki, M. (Centro Medical Nacional, Instituto Mexican del Seguro Social, Av. Cuahtemoc 330, Mexico 7, D.F.). *Arch. Invest. Med. (Mex.)* 8(1):71-83; 1977.

0786 ANATOMY OF THE ANORECTAL SPHINCTER AND ITS SURGICAL IMPLICATIONS. (Por.) Matos, D.; Werneck Lima, J.; De Oliveira, E. (Hospital Sao Paulo, Sao Paulo, Brazil). *Bol. Sanat. Sao Lucas* 38(4):147-160; 1977.

0787 THE EFFECT OF ETHANOL ON THE ULTRASTRUCTURE OF ACINI OF THE PANCREAS. (Rus.) Podolsky, A. E. (N. V. Sklifovsky Res. Inst. Emergency Aid, Moscow, USSR). *Biull. Eksp. Biol. Med.* 83(3):377-379; 1977.

0788 STRUCTURE AND INNERVATION OF THE CHOLEDOCHO DUODENAL JUNCTION. (Eng.) Kyosola, K. (Dept. Anatomy, Univ. Helsinki, Helsinki, Finland). *Ann. Chir. Gynaecol.* 65(Suppl. 192):1-135; 1976.

0789 ENDOCRINE CELLS IN THE NORMAL HUMAN COLON AND RECTUM: ULTRASTRUCTURAL STUDY [Abstract]. (Eng.) Cristina, M. L.; Lehy, T.; Zeitoun, P. (INSERM U-10, Hopital Bichat, 75877 Paris, Cedex 18, France). *Digestion* 16(1/2): 207-208; 1977.

See also, 0795, 0840, 0864, 0878, 0890, 0928, 0967, 1001, 1007, 1008, 1039, 1041, 1053, 1069, 1070, 1113, 1169, 1208, 1210, 1214, 1216, 1266, 1329, 1347, 1561, 1676.

ABSORPTION - EXCRETION - TRANSPORT

0790 POSTPRANDIAL SERUM BILE ACIDS IN HEALTHY MAN: EVIDENCE FOR DIFFERENCES IN ABSORPTIVE PATTERN BETWEEN INDIVIDUAL BILE ACIDS. (Eng.) Angelin, B.; Bjorkhem, I. (Dept. Medicine, Serafimerlasarettet, S-112 83 Stockholm, Sweden). *Gut* 18(8):606-609; 1977.

The hypothesis was tested that substantial dihydroxy bile acid absorption in the proximal small intestine is responsible for observed increases in the cholic acid (C)/chenodeoxycholic acid (CD) and C/deoxycholic acid (D) ratios in intestinal aspirates along the proximal intestine. The serum concentrations of C, CD, and D before and after a standardized meal were determined in five healthy women using a highly specific and accurate gas chromatographic-mass spectrometric technique. The C level rose significantly 60 min after the meal, reached a peak after 90 min, and had returned to the original level after 150 min. In contrast, the serum concentrations of CD and D displayed a significant rise by 30 min, reached a peak after 90 min, but had not returned to fasting levels after 150 min. The results suggest that there is considerable absorption of dihydroxy bile acids in the proximal small intestine in man.

0791 TRANSPORT OF α-AMYLASE ACROSS THE BASOLATERAL MEMBRANE OF THE PANCREATIC ACINAR CELL. (Eng.) Isenman, L. D.; Rothman, S. S. (Dept. Physiology, Univ. California, San Francisco, CA 94143). *Proc. Natl. Acad. Sci.* 74(9): 4068-4072; 1977.

The flux of α-amylase (1,4-α-D-glucan glucanohydrolase; EC 3.2.1.1) across the basolateral membrane of the acinar cell was measured in the cell-to-bath direction using the whole rabbit pancreas in organ culture. This *in vitro* preparation is polarized so that apical and basolateral secretions can be collected separately. The unstimulated amylase flux from cell to bath was substantial at the initial rate (approximately 3 times the concurrent apical flux). With time, bath amylase approached a steady-state concentration, suggesting an equilibrating process. During the same time interval, ductal amylase secretion remained constant. At the steady state, the amylase concentration in the bath was at least an order of magnitude less than its ductal concentration. Hourly replacement of bathing medium reproduced the initial rate of amylase release into the bath for five consecutive hours. Pancreozymin (cholecystokinin) did not alter the steady-state bath amylase content, although it greatly augmented ductal amylase secretion. In contrast, acetyl-β-methyl-choline chloride greatly increased both the flux from the cell to bath and the ductal secretion of amylase. The results indicate a natural bidirectional permeability of the basolateral membrane to digestive enzyme. The results also support evidence previously obtained, which suggested that such a permeability might exist and that these fluxes are all related by a cytoplasmic pool (or pools) of digestive enzyme that acts as both a source of and a repository for enzyme crossing the membrane.

0792 ABSORPTION OF HORSERADISH PEROXIDASE BY NEONATAL PIG INTESTINAL EPITHELIUM. EFFECT OF *Escherichia coli* (O55B5) ON ABSORPTION. (Eng.) Staley, T. E. (Coll. Veterinary Medicine, Stillwater, OK 74074). *Am. J. Vet. Res.* 38(9): 1307-1314; 1977.

The pathway of macromolecular transport through the neonatal pig small intestinal epithelium following the adherence of *Escherichia coli* to the apical ends of enterocytes (monocontamination) was examined and compared with transport in unexposed (sterile) controls, utilizing the cytochemical marker horseradish peroxidase (HRP). In control animals, the marker was adsorbed to apical microvilli inside apical tubules and filling cytoplasmic vacuoles of the enterocytes. Extracellular absorbed marker assumed a spherical appearance within the subepithelial spaces but was dispersed within the capillary lumens. Uptake of HRP into the enterocyte occurred in 48-hr-old neonatal pigs, but transport into the circulation was not observed. Exposure to *E. coli* was accomplished by the intragastric administration of about 1 g of organisms/kg body weight. Adherence of *E. coli* to the surface of the ileal enterocyte did not totally inhibit HRP uptake. The *E. coli* that ad-

hered to the surface of the enterocyte or that were seen within intercellular vacuoles appeared to be static since they were not involved in the transepithelial migration of envacuolated HRP.

0793 STUDIES ON TRANSLOCATION OF IMMUNOGLOBULINS ACROSS INTESTINAL EPITHELIUM. I. IMPROVEMENTS IN THE PEROXIDASE-LABELED ANTIBODY METHOD FOR APPLICATION TO STUDY OF HUMAN INTESTINAL MUCOSA. (Eng.) Isobe, Y.; Chen, S. T.; Nakane, P. K.; Brown, W. R. (Denver Veterans Admin. Hosp., 1055 Clermont St., Denver, CO 80220). *Acta Histochem. Cytochem.* 10(2):161-171; 1977.

The peroxidase-labeled antibody method was adapted for the localization of immunoglobulins and the secretory component in human intestinal mucosa by light and electron microscopy. Modifications of the method included (1) the use of digitonin in the first wash step of tissues fixed in periodate-lysine-paraformaldehyde, (2) double coating of glass slides with gelatin and egg albumin before application of cryostat sections, and (3) inactivation of endogenous peroxidase by periodic acid treatment of cryostat sections. With these improvements, tissue morphology was well preserved, antigenicity of immunoglobulins and the secretory component was retained, penetration of labeled antibodies into cellular compartments was complete, and confusion at the light microscopic level between peroxidase-containing cells and cells stained with peroxidase-labeled antibodies was avoided.

0794 EFFECTS OF 1α,25-,24*R*,25- AND 1α,24*R*,25-HYDROXYLATED METABOLITES OF VITAMIN D_3 ON CALCIUM AND PHOSPHATE ABSORPTION BY DUODENUM FROM INTACT AND NEPHRECTOMIZED RATS. (Eng.) Walling, M. W.; Hartenbower, D. L.; Coburn, J. W.; Norman, A. W. (Veterans Admin. Wadsworth Hosp. Center, Riverside, CA 92502). *Arch. Biochem. Biophys.* 182(1):251-257; 1977.

The effects of 1α,25-dihydroxyvitamin D_3, 24*R*,25-dihydroxyvitamin D_3, and 1α,24*R*,25-trihydroxyvitamin D_3 on active calcium and phosphate transport by rat duodenum were studied in sham-operated and bilaterally nephrectomized vitamin D-deficient rats. Both 1α,25-dihydroxy- and 1α,24*R*,25-trihydroxyvitamin D_3 markedly stimulated calcium and phosphate absorption with similar effects in sham-operated and nephrectomized rats. A 10-fold higher dose of 24*R*,25-dihydroxyvitamin D_3 was required for an equivalent stimulation of absorption in sham-operated rats, and this compound had no effect on duodena from nephrectomized rats. These data provide the first evidence that 24*R*,25-dihydroxy- and 1α,24*R*,25-trihydroxyvitamin D_3 can stimulate the active intestinal absorption of phosphate. The lack of response to 24*R*,25-dihydroxyvitamin D_3 in nephrectomized rats confirms prior results that indicated that renal metabolism of this secosteroid to 1α,24,25-trihydroxyvitamin D_3 is required for biological activity. These experiments provide a simple bioassay technique that apparently reflects the changes in duodenal calcium and phosphate absorption that occur under more rigorous short-circuited conditions and, in particular, can be used for screening putative 1α-hydroxyl analogs of vitamin D in nephrectomized rats.

0795 INTRACELLULAR IRON TRANSPORT IN RAT INTESTINAL EPITHELIUM: BIOCHEMICAL AND ULTRASTRUCTURAL OBSERVATIONS. (Eng.) Humphrys, J.; Walpole, B.; Worwood, M. (Welsh Natl. Sch. Medicine, Heath Park, Cardiff CF4 4XW, Wales). *Br. J. Haematol.* 36(2):209-217; 1977.

Iron absorption by the intestinal epithelial cells was studied in male Wistar rats, using differential centrifugation of mucosal homogenates and electron microscope autoradiography. The maximum concentration of ^{55}Fe (62% of ^{55}Fe in the homogenate) was found 1 hr after intragastric administration. The maximum concentration of ^{55}Fe was found in the mitochondrial fraction after 18 hr. The mitochondrial, lysosomal, and microsomal fractions were examined by electron microscopy after autoradiography. One hour after the administration of ^{55}Fe, grains were associated with all the cell organelles, but in particular with microsomal vesicles. Eighteen hours after ^{55}Fe administration, the majority of the grains were associated with mitochondria; approximately 3% of the mitochondria contained one or more grains 18 hr after administration, demonstrating that the radioactivity in the mitochondrial fraction was associated with mitochondria and not with the other organelles contaminating this fraction. Sections of small intestine were taken 10, 20, and 30 cm from the pylorus. One hour after ^{55}Fe feeding, grains were found over the brush border, terminal web, and lateral cell membrane in the segment 10 cm from the pylorus. Thirty-two percent of the grains were found in the supranuclear regions of cells but were not associated with any particular membranes. At 20 cm, fewer grains were found, but a higher proportion were seen over the brush border and terminal web and a lower proportion over the lateral cell membrane. At 30 cm even fewer grains were seen, and these were distributed throughout the cell constituents. Eighteen hours after iron administration, few grains were present in the 10-cm segment. More were found in the 20-cm segment, 34% being located over mitochondria. The 30-cm segment showed many grains concentrated over mitochondria, endoplasmic reticulum, and ribosomes. The results suggest that mitochondria play a quantitatively significant role in iron metabolism within the epithelial cell but do not take part in the transport of iron across the cell during iron absorption.

0796 THE AGE-DEPENDENCE OF INTESTINAL ABSORPTION USING D-XYLOSE AS AN EXAMPLE. (Eng.) Heimann, G.; Roth, B.; Gladtke, E. (Children's Hosp., Joseph-Stelzmann-Strasse 9, D-5000 Koln 41, W. Germany). *Eur. J. Pediatr.* 124(4):285-294; 1977.

The effects of age and of changes in intestinal motility on absorption were studied in 40 infants. The serum concentrations of D-xylose were measured

from capillary blood samples up to 300 min after the p.o. administration of D-xylose (0.5 g/kg). The absorption kinetics were calculated by a digital computer program. Infants up to the age of 2 months had lower maximal serum concentrations compared to older infants, because of a larger volume of distribution. The rate constant of invasion (k_1), which serves as a measure for the rate of intestinal absorption, and the rate constant of elimination (k_2) were significantly lower in infants up to the age of 2 months compared to older infants. The quotient of k_1/k_2 was independent of age. The time it took to reach maximal serum concentration was significantly longer in the young infants. The administration of metoclopramide (0.5 mg/kg) simultaneously with D-xylose demonstrated that the slower motility of the gastrointestinal tract in newborns and young infants is not exclusively responsible for the slower rate of D-xylose absorption. Experiments in which 26 infants received D-xylose (0.3 or 0.5 g) suggested that absorption involves saturation kinetics.

0797 ABSORPTION, AND EFFECT ON GASTRIC MUCOSA, OF BUFFERED AND NON-BUFFERED TABLETS OF ACETYLSALICYLIC ACID. (Eng.) Linnoila, M.; Lehtola, J. (Duke Univ. Medical Center, Box 2921, Durham, NC 27710). *Int. J. Clin. Pharmacol. Biopharm.* 15(2):61-64; 1977.

The effects of buffered and nonbuffered acetylsalicylic acid tablets on the gastric mucosa and on gastric distress were investigated in 12 healthy subjects and in 42 rheumatoid arthritic patients, resp. The 12 healthy subjects received either acetylsalicylic acid (500 mg), acetylsalicylic acid buffered with magnesium oxide (175 mg), or acetylsalicylic acid buffered with magnesium oxide (100 mg) and aluminum aminoacetate (75 mg). One hour after the administration of the tablets, the acetylsalicylic acid level in the serum was significantly higher for the nonbuffered preparation than for the buffered preparation. Two hours after administration, this situation was reversed. However, the absolute means at any point in time differed by less than 2 µg. The serum salicylic acid levels were significantly higher 30, 60, and 90 min after the administration of the tablets buffered with magnesium oxide than after the other two types of tablets ($p<0.05$). In six healthy subjects in whom gastroscopy was carried out before and after 8 days of treatment with the preparations, gastric mucosal damage tended to be milder after the preparation buffered with magnesium oxide than after the nonbuffered preparation. Subjective feeling of gastric distress was observed in the 42 arthritic patients as often after buffered tablets as after nonbuffered tablets. These studies show that the absorption of salicylic acid is not affected in clinically significant amounts by buffering the tablets.

0798 THE DISPOSITION OF RADIOACTIVITY AFTER ADMINISTRATION OF THE ANTHELMINTHIC METHYL-^{14}C-5-CYCLOPROPYLCARBONYL-2-BENZIMIDAZOLE CARBAMATE (CICLOBENDAZOLE) TO RATS AND DOGS. (Eng.) Brodie, R. R.; Mayo, B. C.; Chasseaud, L. F.; Hawkins, D. R. (Dept. Metabolism and Pharmacokinetics, Huntingdon Res. Center, Huntingdon, England). *Drug Res.* 27 (3):593-598; 1977.

The disposition of radioactivity was studied in rats and dogs after administration of a new anthelmintic agent, ^{14}C-labeled methyl-5-cyclopropylcarbonyl-2-benzimidazole carbamate (^{14}C-ciclobendazole). After a p.o. dose of ^{14}C-ciclobendazole (4 mg/kg) to rats, about 70% and 20% of the dose was excreted in the feces and urine, resp., during 2 days. Bile duct cannulated rats excreted about 80% of the dose in 48-hr bile, about 2% in the feces, and about 10% in the urine, showing that the p.o. dose was well absorbed and that some enterohepatic circulation probably occurred. The excretion of radioactivity in the bile was less after i.v. administration. A p.o. dose of ^{14}C-ciclobendazole (4 mg/kg) to dogs was mainly eliminated during 2 days with about 80% of the dose in the feces and only about 10% in the urine. Anesthetized bile duct cannulated dogs excreted between 26% and 35% of a p.o. dose in the bile during 24 hr and up to 58% was absorbed at this time. There was no unusual accumulation or localization of radioactivity in the tissues of rats or dogs following single or multiple p.o. doses of the drug. The highest concentrations were present in the intestinal tract, liver, and kidneys. After p.o. administration of ^{14}C-ciclobendazole to rats at three different dose levels (4, 40, and 400 mg/kg), peak plasma levels occurred at 15-30 min and declined with similar half-lives (about 20 hr). Ciclobendazole absorption was probably dose-dependent, a lower proportion probably being absorbed at higher doses. After repeated daily p.o. dosing with ^{14}C-ciclobendazole (4 mg/kg), there were no significant changes in either the daily plasma concentrations or the biological half-life measured after the last dose, indicating that ciclobendazole probably did not induce or inhibit its own metabolism.

0799 ABSORPTION OF ^{14}C-BUMADIZONE-Ca AND ^{14}C-PHENYLBUTAZONE IN ISOLATED INTESTINAL SEGMENTS IN VITRO AND TIED-OFF GASTROINTESTINAL SECTIONS IN VIVO OF RATS AND GUINEA PIGS. (Eng.) Seebald, H.; Forth, W. (Lehrstuhl fur Pharmakologie und Toxikologie, Ruhr Universitat, Postfach 10 21 48, D-4630 Bochum, W. Germany). *Drug Res.* 27(1):624-635; 1977.

The absorption of butylmalonic acid mono(1,2-diphenylhydrazide) (bumadizone) and its condensation product was investigated in intestinal segments of rats and guinea pigs *in vitro* and *in vivo*. ^{14}C-labeled bumadizone-Ca and phenylbutazone were administered. In isolated jejunal segments of rats and guinea pigs *in vitro*, the transfer and the uptake of bumadizone by tissue was proportional to the concentration administered on the mucosal side. Bumadizone (320 nmol/ml) slightly decreased the absorption of water and glucose. Phenylbutazone inhibited the absorption of water and glucose by rat jejunal segments at 174 nmol/ml or greater. Transfer and uptake

of phenylbutazone by tissue was proportional only up to 174 nmol/ml. Beyond this concentration they decreased. In guinea pigs, transfer was only one-fourth, and uptake by tissue only one-half of that in rat jejunal segments. The *in vivo* rat jejunum was found to be the main site of absorption of both bumadizone and phenylbutazone. Absorption was nearly complete within 1 hr after administering a dose of 0.64 µmol. In guinea pigs it was incomplete and was only one-fourth to one-third of that in rats. In the stomach, ileum, and colon, 1.5 to 3 times as much phenylbutazone was absorbed, as compared with bumadizone. Due to higher solubility in aqueous fluids, the availability of bumadizone-Ca for absorption exceeded that of phenylbutazone (1.62-97.2 µmol). In experiments with isolated jejunal segments of rats and guinea pigs *in vitro*, the capability of the intestinal tissue to produce phenylbutazone from bumadizone was demonstrated. Within 2 hr, nearly 20% of the ^{14}C-bumadizone was converted into phenylbutazone in both rat and guinea pig segments after the administration of bumadizone (16 µmol/ml).

0800 QUANTITATIVE STUDIES IN VITRO ON UPTAKE AND ESTERIFICATION OF PALMITATE INTO HUMAN AND RAT JEJUNAL MUCOSA. (Eng.) Sueur, R.; Cerf, M.; Di Costanzo, G.; Debray, C. (Hopital Bichat, Universite Paris VII, 170, Boulevard Ney, F-75877 Paris Cedex 18, France). *Digestion* 15(1): 34-42; 1977.

Quantitative incorporation and esterification of (9-10) ^{3}H-palmitate into human jejunal mucosa was measured *in vitro* to develop a technique for the determination of the esterifying capacity of the human intestinal mucosa sufficiently reliable to be applied semi-routinely in various pathological conditions. The results of the study were compared with results obtained in everted rat intestinal rings. Rat everted jejunal segments and jejunal biopsies from six normal subjects and two patients with adult celiac disease were incubated at 37 C in 4 ml of micellar solution (10 µM/ml sodium taurocholate, 3.6 µM/ml labeled palmitic acid, and 5 µM/ml glucose made up in Krebs-Ringer phosphate buffer, pH 7.4, Ca^{2+} omitted). Palmitate incorporation into diglyceride (DG) in the rat was higher than that into triglyceride (TG) with a TG:DG ratio lower than 1. Incorporation into cholesterol esters (CE) and phospholipid (PL) was low. There was a low but significant incorporation into monoglyceride (MG). Free fatty acid (FFA) uptake was high. In human jejunum, palmitate incorporation into CE, PL, and MG was low, reaching apparently a steady state by 5 min of incubation. Incorporation into TG was high, with a TG:DG ratio of about 3:1. Uptake into FFA was lower in man than in the rat, although the dispersion of the results was great. In the patients with celiac disease, the uptake into FFA was similar to that in the normal subjects. The incorporation of palmitate into TG and DG was increased with a TG:DG ratio below 2:1. The incorporation into TG was increased by the addition of monoolein into the incubation medium, but the total palmitate uptake was unchanged.

0801 ABSORPTION OF PHARMACOLOGIC DOSES OF ZINC. (Eng.) Oelshlegel, F. J.; Brewer, G. J. (Univ. Mississippi Medical Center, Jackson, MS 39216). *Prog. Clin. Biol. Res.* 14:299-311; 1977.

Absorption of orally administered zinc in the form of zinc sulfate was studied in fasting normal subjects. Assays of blood taken 0-4 hr after a 110-mg dose of zinc sulfate (providing 25 mg zinc) demonstrated that zinc absorption varies greatly in individuals. Varying the doses from 12.5 mg to 50 mg of zinc demonstrated that the smaller dose is more efficiently absorbed. Also, to maintain elevated plasma levels of zinc, doses of zinc should not be administered more than 4 hr apart. Since zinc sulfate is not well tolerated, zinc was also administered in the form of acetate and carbonate salts. Plasma levels of zinc were identical after administration of 25 mg zinc as the acetate or as the sulfate, but significantly lower ($p<0.05$) after the carbonate than after the sulfate. The effect of a number of foods (including hamburger on a bun, celery, lemon, coffee, and milk) ingested with the zinc sulfate on plasma zinc levels was studied. Most foods inhibited zinc absorption with the exception of bacon, hamburger meat, and, possibly, soft drinks, tea, and apples. Apparently, many dietary components such as fiber, phytic acid, and calcium block zinc absorption and may lead to zinc deficiencies.

0802 WEAK ELECTROLYTE TRANSFER IN THE GUINEA PIG JEJUNUM: SECRETION OF TRIMETHOXYBENZOIC ACID. (Eng.) Kolassa, N.; Krivanek, P.; Turnheim, K. (Pharmakologisches Institut, Universitat Wien, Wahringerstrasse 13a, A-1090 Wien, Austria). *Naunyn Schmiedebergs Arch. Pharmacol.* 298(3):223-228; 1977.

0803 FURTHER STUDIES ON THE MECHANISM OF THE ABSORPTION OF ION PAIR COMPLEX FROM THE RAT SMALL INTESTINE. (Eng.) Nakamura, J.; Muranushi, N.; Kimura, T.; Muranishi, S.; Sezaki, H. (Faculty Pharmaceutical Sciences, Kyoto Univ., Yoshida Shimoadachi-cho, Sakyo-ku, Kyoto, Japan). *Chem. Pharm. Bull.* 25(5):851-858; 1977.

0804 NORMAL ABSORPTION. (Eng.) Sleisenger, M. H.; Brandborg, L. L. *In: Malabsorption.* Edited by L. H. Smith, Jr. (Philadelphia: W. B. Saunders Co.): 13:2-50; 1977.

0805 ABSORPTION OF VITAMIN B_{12} AND IRON. (Eng.) Callender, S. T. *In: Topics in Gastroenterology.* Edited by S. C. Truelove and J. A. Ritchie. (Oxford: Blackwell Scientific Publications): (4):163-172; 1976.

0806 IN VIVO ACTIVATION ANALYSIS. (Eng.) Boddy, K. *In: Topics in Gastroenterology.* Edited by S. C. Truelove and J. A. Ritchie. (Oxford: Blackwell Scientific Publications): (4):273-288; 1976.

0807 RESORPTION OF LIPASE OF PANCREATIC ORIGIN FROM INTACT, FULLY DEVELOPED INTESTINE INTO THE DUODENAL LYMPH NODES. (Hun.) Papp, M.; Feher, S.; Folly, G.; Horvath, J.; Ormai, S. (Magyar Tudomanyos Akademia Kiserleti Orvostudomanyi Kutato Intezete, Hungary). *Orv. Hetil.* 118(24):1391-1393; 1977.

0808 HARMALINE, AN INHIBITOR OF INTESTINAL TRANSPORT PROCESSES ASSOCIATED WITH THE MIGRATION OF SODIUM IONS. (Fre.) Sepulveda, F. V.; Buclon, M.; Robinson, J. W. L. (Centre Hospitalier Universitaire Vaudois, 1011 Lausanne, Switzerland). *Gastroenterol. Clin. Biol.* 1(1):87-93; 1977.

0809 INVESTIGATIONS OF THE BIOAVAILABILITY OF IRON FROM BI- AND TRIVALENT IRON SALTS. (Ger.) Dietzfelbinger, H.; Kaboth, W. (Krankenhauses Munchen-Schwabing, Kolner Platz 1, D-8000 Munich 40, W. Germany). *Med. Klin.* 72(15):654-659; 1977.

0810 CHRONOBIOLOGICAL ASPECTS OF IRON METABOLISM. CLINICAL-THERAPEUTIC IMPLICATIONS. (Ita.) Tarquini, B. (Instituto di Semeiotica Medica, Universita, Florence, Italy). *Recent. Prog. Med. (Roma)* 62(2):159-180; 1977.

0811 METABOLISM AND PHARMOKINETICS OF PIPROZOLIN IN RAT, DOG AND MAN. FIRST COMMUNICATION: INVESTIGATIONS WITH THE RADIOACTIVE SUBSTANCE. (Ger.) Vollmer, K. O.; Hodenberg, A. V.; Poisson, A.; Bode, R.; Schauerte, E. (Godecke AG, Abteilung Biochemie, Mooswaldallee, 7800 Freiburg/Brsg., W. Germany). *Arzneim. Forsch.* 27(I, 2b):502-508; 1977.

0812 INHIBITION BY POLYVINYLPYRROLIDONE (PVP) OF GASTROINTESTINAL ABSORPTION OF SALICYCLIC ACID AND RELATED COMPOUNDS IN RATS AND ITS MECHANISM FROM THE POINT OF INTERMOLECULAR INTERACTION. (Jpn.) Okano, T.; Watanabe, Y.; Yoshimura, K. (Pharmaceutical Inst., Tohoku Univ., Aobayama, Sendai, 980, Japan). *J. Pharm. Soc. Jpn.* 96(12): 1432-1439; 1976.

0813 ABSORPTION OF CARBOHYDRATES AND LIPIDS IN THE ALLOGENEIC SMALL INTESTINE. (Rus.) Vinogradova, M. A.; Kirpatovsky, I. D.; Panichenko, V. F.; Lysenko, A. I. (Central Res. Inst. Gastroenterology, Patrice Lumumba Peoples' Friendship Univ., Moscow, USSR). *Biull. Eksp. Biol. Med.* 83(2): 153-156; 1977.

0814 EFFICACY AND ADAPTATION TO FOOD QUALITY OF STARCH-GLUCOSE CONVEYOR AFTER BILE-PANCREATIC DUCT LIGATION IN RATS. (Rus.) Loginov, G. I. (Andizhan Medical Inst., Andizhan, USSR). *Biull. Eksp. Biol. Med.* 83(4):406-408; 1977.

0815 MECHANISMS OF pH ADJUSTMENT BY THE JEJUNUM IN DOGS. INFLUENCES ON WATER, NaCl AND GLUCOSE ABSORPTION. (Fre.) Dolisi, C.; Ozon, C.; Soummer, A. M.; Ardisson, J. L. (Laboratoire de Physiologie, Faculte de Medecine, Avenue de Vallombrose, F 06034 Nice Cedex, France). *J. Physiol. (Paris)* 72(7):893-902; 1976.

0816 THE ENTEROCYTIC PHASE OF THE ABSORPTION OF TRIGLYCERIDES. (Fre.) Theodore, C.; Cerf, M. (Hopital Bichat, 170 boulevard Ney, F 75877 Paris Cedex 18, France). *Gastroenterol. Clin. Biol.* 1(4): 369-376; 1977.

0817 EXPLORATION OF INTESTINAL ABSORPTION. CRITICAL ASSESSMENT OF THE D-XYLOSE TEST. (Spa.) Aparisi-Quereda, L.; Rodrigo-Gomez, J. M.; Guix-Garcia, J.; Serra-Desfilis, M. A.; Del Olmo Puchalt, J. A.; Wassel, A. H. (Catedra de Patologia y Clinica Medicas "B", Facultad de Medicina, Universidad de Valencia, Valencia, Spain). *Rev. Esp. Enferm. Apar. Dig.* 50(2):197-230; 1977.

0818 STUDY OF DIGESTIVE ABSORPTION IN GASTRECTOMIZED DOGS WITH AN INVERTED INTESTINAL SEGMENT. (Spa.) Rodriguez Montes, J. A.; Murillo, A.; Argelus, I. (Catedra I de Patologia y Clinica Quirurgicas, Universidad de Granada, Granada, Spain). *Rev. Esp. Enferm. Apar. Dig.* 50(2):123-128; 1977.

See also, 0823, 0843, 1000, 1016, 1033, 1035, 1070, 1235, 1252, 1627, 1632, 1637, 1661.

MOTILITY

0819 ROLE OF GASTRIN AND OTHER GASTROINTESTINAL HORMONES IN THE PATHOPHYSIOLOGY OF ACHALASIA OF THE ESOPHAGUS. (Eng.) Santoki, O. (Yamaguchi Univ. Sch. Medicine, Ube, Yamaguchi, 755, Japan). *Arch. Jpn. Chir.* 46(2):88-112; 1977.

The effects of gastrin, secretin, cholecystokinin-pancreozymin (CCK-PZ), and Mecholyl on the lower esophageal sphincter pressure (LESP) were investigated in normal dogs, dogs with experimental achalasia, and in seven patients with achalasia. Achalasia was produced in the dogs by the injection of a 5% phenol solution into the muscular layers of the lower esophagus. The i.v. administration of

tetragastrin (0.5, 1.0, 2.0, 5.0, 7.5, and 10.0 μg/kg) in these dogs produced a dose-dependent increase in the LESP; this curve shifted to the left in comparison with that in normal dogs, with each dose of tetragastrin. After the administration of 5.0 μg/kg of tetragastrin in 10 dogs with achalasia and 9 normal dogs, the LESP increased from a mean resting pressure of 8.4 ± 0.50 cm H_2O to 65.0 ± 3.67 cm H_2O in the dogs with achalasia, while that in the normal dogs increased from a resting pressure of 7.0 ± 0.83 cm H_2O to 40.1 ± 2.33 cm H_2O. The pressure change in response to tetragastrin in the dogs with achalasia was thus significantly greater than that in the normal dogs ($p<0.01$). The i.v. injection of secretin (5 CHRU/kg/5 min) had no significant effect on the resting LESP in normal and dogs with achalasia. The increase in LESP seen after tetragastrin was inhibited by a background dose of secretin in both normal and in dogs with achalasia. However, the percent reduction in the dogs with achalasia was not significantly different from that in the normal dogs, suggesting that the effect of secretin was not related to the destruction or degeneration of Auerbach's plexus. The injection of CCK-PZ (2.5 CHRU/kg, i.v.) increased the LESP in both normal and dogs with achalasia, and the response was significantly greater in the latter. The i.m. injection of Mecholyl (10 mg) produced an increase of 29.3 ± 2.75 cm H_2O in the normal dogs and an increase of 72.4 ± 3.80 cm H_2O in the dogs with achalasia ($p<0.01$). The gastrin levels were high in dogs treated with Mecholyl, suggesting that the LESP response may be partially mediated by the release of endogenous gastrin. In the human patients, the LESP increased significantly in response to 5 μg/kg of tetragastrin. This finding indicates that the LESP response to tetragastrin is useful in the preoperative diagnosis of achalasia.

0820 THE EFFECTS OF TRANSECTIONS ON THE BASIC ELECTRICAL RHYTHM IN THE CANINE JEJUNUM. (Eng.) Mendel, C.; Pousse, A.; Dauchel, J.; Schang, J. C.; Grentier, J. F. (Unite de Recherche 61 de l'INSERM, Strasbourg-Hautepierre, France). *Eur. Surg. Res.* 8(6):528-535; 1976.

The effects of transection on the electrophysiology of the canine jejunum were studied. The basic electrical rhythm (BER) was studied *in situ* in 14 anesthetized dogs by the means of six monopolar electrodes. The BER frequency was measured in the intact intestine and after proximal and distal transections of the gut 5 cm apart from the electrodes. No significant differences were found between the BER frequencies recorded by each electrode, demonstrating the existence of a frequency plateau that remained after each transection. In the intact intestine the frequency in the plateau was 13.2 ± 0.3 cycles per minute (cpm). After the proximal transection, this frequency dropped to 12.0 ± 0.4 cpm ($p<0.05$) and, after the distal transection, it dropped to 10.9 ± 0.4 cpm ($p<0.05$). This last frequency change shows the influence of the distal intestine on the electrical behavior of the jejunum.

0821 GASTRIC EMPTYING AND SECRETION IN THE RHESUS MONKEY. (Eng.) Dubois, A.; Natelson, B. H.; van Eerdewegh, P.; Gardner, J. D. (Natl. Inst. Arthritis, Metabolism, and Digestive Diseases, Bethesda, MD 20014). *Am. J. Physiol.* 232(2):E186-E192; 1977.

The volume and composition of the gastric contents as well as the rates of gastric emptying and gastric secretion were studied in the intact stomachs of eight conscious chair-adapted rhesus monkeys after fasting and after a liquid meal to investigate the physiologic variables that regulate gastric emptying and gastric secretion. Esophagotomies were prepared after 3 weeks of chair-adaptation, and experiments were begun 2-3 weeks later. Liquid meals (100 ml) were administered through a double-lumen sump tube passed into the stomach. Periodic aspiration of the gastric contents was carried out to measure gastric secretion by determining the rate of dilution of phenol red administered either separately after an overnight fast (5 ml, 280 mg/l, pH 7.4) or with the meal (40 mg/l, pH 7.4). Gastric emptying was similarly measured by the change in volume of the gastric contents. The mean intragastric volume during fasting was 8 ± 1 ml, and the fractional rate of emptying was 3.9 ± 0.6%/min or 0.31 ± 0.07 ml/min; the rate of water secretion was 0.33 ± 0.03 ml/min. The rates of ion secretion for hydrogen, sodium, potassium, and chloride were 29 ± 4, 18 ± 4, 8 ± 1, and 54 ± 9 μEq/min, resp. Instillation of a meal caused the fractional rate of emptying to increase rapidly to 17.6 ± 3.3%/min ($p<0.01$), return to the fasting level 10-40 min later, and then to increase again to 11.4 ± 2.9%/min ($p<0.02$). The rate of water secretion, following a similar time course, increased rapidly to 4.0 ± 0.6 ml/min ($p<0.001$), decreased to the fasting level 20-30 min later, and again increased significantly ($p<0.02$) 40-50 min after the meal. The hydrogen ion secretion showed a significant increase ($p<0.01$) approximately 10-30 min after the meal, as did the chloride secretion, which rose to 95-110 μEq/min. Potassium secretion increased to 12-18 μEq/min ($p<0.05$). The results demonstrate the complexity of gastric response to meal-stimulation and comprise the first study in which gastric emptying and secretion are determined simultaneously in conscious chair-adapted primates.

0822 HUMAN LOWER ESOPHAGEAL SPHINCTER (LES) RESPONSE TO SUBMAXIMAL AND MAXIMAL EFFECTIVE DOSES OF SYNTHETIC HUMAN GASTRIN I (G-17) IS NOT INHIBITED BY ATROPINE [Abstract]. (Eng.) Jensen, D. M.; McCallum, R. W.; Walsh, J. H. (Veterans Admin. Wadsworth Hosp. Center, Los Angeles, CA). *Gastroenterology* 72(5, Part 2):1076; 1977.

0823 RELATIONSHIP BETWEEN INTESTINAL MOTILITY AND INTESTINAL ABSORPTION. (Fre.) Matuchansky, C.; Rambaud, J. C.; Modigliani, R.; Bernier, J. J. (Service de Gastroenterologie, Hotel-Dieu,

F 86021 Poitiers, France). *Gastroenterol. Clin. Biol.* 1(3):225-229; 1977.

0824 ROLE OF THE DUODENUM AND ANTRUM IN THE REGULATION OF THE PRESSURE OF THE LOWER ESOPHAGEAL SPHINCTER IN THE DOG. (Ger.) Sauberli, H.; Largiader, F.; Koelz, H. R.; Hollinger, A.; Weber, A.; Siewert, R.; Blum, A. L. (Chirurgische Universitatsklinik A, Ramistrasse 100, CH-8091 Zurich, Switzerland). *Helv. Chir. Acta* 43(5/6): 799-802; 1976.

0825 EXPERIMENTAL IMPROVED MODEL OF SMALL INTESTINAL ILEUS IN RABBIT AND A TRIAL OF ITS USE FOR MEASURING THE DURATION OF POSTOPERATIVE INTESTINAL PARALYSIS. (Pol.) Kurzbauer, R. (Z. Oddzialu Chirurgicznego Zespolu Opieki Zdrowotnej w Prudniku, Prudnik, Poland). *Pol. Przegl. Chir.* 49(4):419-423; 1977.

0826 MOTILITY OF THE COLON. (Ger.) Wienbeck, M. (2. Medizin. Klinik und Poliklinik, Moorenstrasse 5, 4000 Dusseldorf 1, W. Germany). *Z. Gastroenterol.* 15(3):209-214; 1977.

0827 GASTRIC EMPTYING. (Ger.) Guller, R. (Kantonsspital Basel, Hebelstr. 1, CH-4056 Basel, Switzerland). *Z. Gastroenterol.* 15(3):185-191; 1977.

0828 GASTRIC MOTILITY AND LYSPAFEN® (PENTAPIPERIDE). (Ger.) Kimmig, J. M.; Heinkel, K.; Ensslin, H. (Medizinische Klink, Krankenhaus Stuttgart-Bad Cannstatt, Priessnitzweg 24, D-7000 Stuttgart 50, W. Germany). *Z. Gastroenterol.* 14(5):531-535; 1976.

0829 RESTORATION OF THE ELECTRICAL ACTIVITY AND INTRAMURAL NERVE APPARATUS AFTER STOMACH WALL TRANSECTION IN THE DOG. (Rus.) Solovieva, I. A.; Atanassova, E. (Pavlov's Inst. Physiology, Acad. Sciences USSR, Leningrad, USSR). *Fiziol. Zh. SSSR* 63(5):723-734; 1977.

0830 PYLORIC SPHINCTER DISCRIMINATING ABILITY WITH RESPECT TO SOLID PARTICLES OF THE GASTRIC CONTENT. (Ukr.) Kharchenko, N. M. (Inst. Physiology, Kiev Univ., Kiev, USSR). *Fiziol. Zh.* 22(4):505-509; 1976.

0831 PERIPHERAL MECHANISMS OF THE SMALL INTESTINE CAPACITANCE VESSEL RESPONSES TO NORADRENALIN. (Rus.) Kudriashov, Yu. A. (Inst. Experimental Medicine, Acad. Medical Science USSR, Leningrad, USSR). *Fiziol. Zh. SSSR* 63(4): 557-564; 1977.

See also, 0788, 0834, 0855, 1062, 1071, 1098, 1105, 1161, 1199, 1280, 1282, 1637, 1673.

SECRETION AND METABOLISM

0832 ACTION OF TESTOSTERONE ON THE DIFFERENTIATION AND SECRETORY ACTIVITY OF A TARGET ORGAN: THE SUBMAXILLARY GLAND OF THE MOUSE. (Eng.) Chretien, M. (Departement de Biologie, Centre d'Etudes Nucleaires de Saclay, Gif-sur-Yvette, France). *Int. Rev. Cytol.* 50:333-396; 1977.

0833 SALIVARY GLAND GLUCAGON: EFFECT OF AGE AND SEX [Abstract]. (Eng.) Kelly, J.; Paloyan, V.; Edwards, K.; Kisla, J.; Hojvat, S.; Lawrence, A. M. (Veterans Admin. Hosp., Hines, IL). *Clin. Res.* 25(4):572A; 1977.

0834 CAERULEIN (CERULETIDE). A REVIEW. (Eng.) Ganzina, F.; Santamaria, A. (Ric. Med. Farmitalia, Milan, Italy). *Acta Gastroenterol.* 39(5/6):169-185; 1976.

0835 ROLE OF THE KIDNEY IN GASTRIN METABOLISM. (Eng.) Yamagata, S.; Ishimori, A.; Sakurada, H.; *et al.* (Tohoku Univ., Sch. Medicine, Sendai, Japan). *Tohoku J. Exp. Med.* 118(Suppl.): 45-51; 1976.

See also, 1055, 1056, 1064, 1070.

SECRETION AND METABOLISM

Stomach

0836 METABOLIC MEMBRANE ASPECTS OF GASTRIC H^+ TRANSPORT. (Eng.) Sachs, G.; Chang, H.; Rabon, E.; Shackman, R.; Sarau, H. M.; Saccomani, G. (Univ. Alabama Medical Sch., University Station, Birmingham, AL 35294). *Gastroenterology* 73(4):931-940; 1977.

Information concerning the acid secretory response of the parietal cell to changes of second messengers (i.e., Ca^{2+}, cyclic AMP, or some unknown component) is reviewed. Metabolic properties of dog gastric mucosa, investigated by substrate level measurements, implicate the Krebs cycle as the major energy-yielding metabolic pathway, but are equivocal in terms of an ATP-based H^+ secretion. Purification of gastric membranes by centrifugation and free flow electrophoresis results in a class of membrane vesicles enriched in K^+-ATPase and capable of ATP-energized H^+ uptake. Immunohistochemistry shows these to be derived from the parietal cell. H^+ uptake by the vesicles is accompanied by K+ efflux, and movement of either ion is not potential-coupled. The simplest interpretation of these transport studies is uptake of KCl by the vesicles by passive diffusion followed by active H+:K+ exchange. In some respects, however, this model fails to conform to the expectations from *in vitro* studies. It may be, therefore, that another pump (i.e., redox) or another membrane component (i.e., Cl^- conductance) is lost during purification. The properties of the vesicles are such, however, as to establish their role in H^+ secretion by the stomach.

0837 IN VITRO RELEASE OF GASTRIN FROM ISOLATED PERFUSED ANTRUM. (Eng.) Dretler, R.; Wesdorp, R. I.; Fischer, J. E.; Soeters, P. B.; Ebeid, A. M. (Harvard Medical Sch., Boston, MA 02114). *Am. J. Surg.* 134(2):237-241; 1977.

An isolated rat gastric antrum preparation is described that may be useful in the study of the physiology of gastrin. Gastrin antra were removed from fasting female Sprague-Dawley rats and placed in a 2-ml perfusion chamber. Reservoir bottles containing oxygenated Krebs-Ringer's A solution either as control perfusion solution or with added agent to be tested were connected to the perfusion chamber by tubes that passed through glass-jacketed heat exchange systems. Flow rate was regulated by a perfusion pump, usually at the rate of 30 ml/hr. Gastrin secretion was measured by radioimmunoassay after perfusion of the gastric antra with 50 mM acetylcholine (+ 0.1 mM eserine to prevent destruction of acetylcholine by cholinesterase), 50 mM glycine, or 30 mM or 10 mM calcium chloride added to the perfusion solution. Gastrin secretion showed a biphasic response to stimulation by acetylcholine and a single phase response to glycine and calcium chloride stimulation. Gastrin secretion from minced rather than intact antra perfused with acetylcholine showed a similar biphasic response. The biphasic release of gastrin after acetylcholine administration suggests that antral gastrin may exist in two cell populations, one of which is more accessible than the other. It is concluded that the described preparation simplifies the study of antral gastrin release, eliminating many of the inhibitory feedback mechanisms.

0838 INHIBITION OF GASTRIN RELEASE AND GASTRIC SECRETION OF ACID. (Eng.) Stadil, F.; Rehfeld, J. F.; Christensen, K. C.; Christiansen, J.; Dano, P.; Stage, J. G. (Rigshospitalet, 2100 Copenhagen, Denmark). *Scand. J. Gastroenterol.* 11(42):25-35; 1977.

Recent studies on gastrin that are relevant to the inhibition of gastric acid secretion are summarized to provide a better understanding of the physiologic mechanisms involved. A number of physiologic systems for nervous and humoral inhibition of the secretion of gastric acid and release of gastrin are discussed. Vagal activation of the stomach, which may be cholinergic or noncholinergic, has been described in relation to the effects of epinephrine, norepinephrine, beta-adrenergic blockade, and vagotomy. The effect of diverting the gastric juice from the stomach during insulin stimulation and continuous aspiration of gastric juice are discussed. Intestinal influences, both stimulatory and inhibitory, on gastric acid secretion are presented. The intestinal actions were studied by experiments with intestinal bypass operations and on the effects of secretin, fat, and glucose administration. Intestinal inhibitors are the more important factors in gastric acid secretion and gastrin release. It is suggested that identification of these humoral inhibitors may give a better understanding of the physiologic mechanism involved.

0839 ACID HYDROLASES: ASSAY OF ACTIVITY AND LATENCY IN THE VARIED MIXED CELL POPULATIONS OF CANINE GASTRIC MUCOSA. (Eng.) Boutros, M. I. R.; Waldron-Edward, D. (Div. Surgical Res., McGill Univ., Montreal, Quebec H3C 3G1, Canada). *Lab. Invest.* 36(4):436-443; 1977.

The specific enzymic properties, membrane or particle binding capacities, and the total activities of acid hydrolases were compared in normal canine antral and fundic mucosae and in liver. Basing each level of activity on tissue content of either protein or DNA, most of the enzymes were higher in the antrum than in the fundus, indicating that the normal, internal "digestive" or autophagic processes of the antral mucosa, with its preponderance of mucin-producing cells, are more pronounced than in the main body of the gastric mucosa. Acid phosphatase, α-galactosidase, β-galactosidase, and α-fucosidase showed differences in pH profile. In both fundic and antral tissues, acid phosphatase, although sharing the same peak of maximal activity at pH 4.8, showed a broad band of high activity in the fundic mucosa. An extra acidic peak of α-fucosidase at pH 3.3 was found in the antrum only. The specific activities of acid *p*-nitrophenylphosphatase, β-*N*-acetylglucosaminidase, α-galactosidase, arylsulfatase, α-fucosidase, and cathepsin D in the antrum (29 ± 14, 35 ± 2, 1.0 ± 0.3, 6.0 ± 0.5, 4.5 ± 0.8, and 357 ± 99 nmol/min/mg protein, resp.) were higher than those in the liver (20 ± 2, 19 ± 1, 0.39 ± 0.11, 2.7 ± 0.2, and 33 ± 2, resp., with no value given for arylsulfatase), whereas the activities of antral β-*N*-acetylgalactosaminidase, β-galactosidase, and mitochondrial succinate dehydrogenase were lower (2.1 ± 0.4 versus 2.8 ± 0.2, 2.1 ± 0.2 versus 2.9 ± 0.2, and 5.3 ± 1.6 versus 23 ± 5 nmol/min/mg, resp.). The fundic mucosa showed enzyme levels close to those of the liver with the excep-

tion of α-fucosidase, which was significantly lower than that in the liver. The high specific activity of cathepsin D in both the antrum and fundus, compared with that in the liver, suggests that the lysosomal enzymes in the canine mucosa may have a special function.

0840 CYCLIC AMP AND CYCLIC GMP IN THE RAT GASTRIC MUCOSA: THE SIGNIFICANCE OF POST MORTEM CHANGES AND TISSUE FIXATION ON THE ESTIMATION OF THE IN VIVO LEVEL. (Eng.) Ruoff, H. J. (Pharmakologisches Institut der Universitat Tubingen, Wilhelmstrasse 56, D-7400 Tubingen, W. Germany). *Naunyn Schmiedebergs Arch. Pharmacol.* 298(2):167-173; 1977.

The effects of tissue fixation and post-mortem changes on estimations of the *in vivo* levels of cyclic (c)AMP and cGMP (guanosine monophosphate) in the gastric mucosa of the rat were investigated. The tissue was fixed with microwave (MW) irradiation (2.1 kW, 2.45 megaHz), and the influence of delayed inactivation of the mucosa on the activities of adenyl cyclase (AC) and phosphodiesterase (PDE), on the levels of cAMP and cGMP, and the effects of various drugs on the levels of cAMP and cGMP were investigated in 24-hr fasted female FW49 Biberach rats. MW irradiation rapidly inactivated AC and PDE activities; 75% and 0% of AC activity remained after 5 and 25 sec, resp., and 9% and 0% of PDE activity remained after 25 and 45 sec, resp. In rats killed by decapitation, the gastric mucosal cAMP concentration doubled within 2 min after death, and the cGMP level at first (5 sec) dropped by 50% and then rose to nearly the initial concentration; 50-sec exposure of living rats to MW irradiation prevented the change in cAMP concentration, but shorter MW exposures did not. Degradation of cAMP by the MW exposure did not occur. In rats killed by decapitation and then exposed to MW for 50 sec at 5, 10, 20, 30, 60, 120, and 480 sec post-mortem, the changes in cAMP and cGMP levels were similar to those in the decapitated rats not exposed to MW, the cAMP level at 2 min, 4 min, and 8 min being significantly elevated ($p<0.001$, $p<0.001$, $p<0.005$, resp.) and the cGMP concentration at 5 sec being significantly ($p<0.01$) decreased. Hence, although MW irradiation killed the rats in 1-2 sec, longer exposures were required for mucosal fixation. Therefore, measurements made after 50-sec exposures of living rats to MWs were defined as *in vivo* levels, and measurements made in tissue fixed by 50-sec MW treatment applied 2 min after decapitation were defined as post mortem levels. Theophylline (0-200 mg/kg, i.p.) increased the *in vivo* cAMP and cGMP levels and the post-mortem cAMP level markedly and in a dose dependent manner. The post-mortem cGMP level was increased only by the highest dose ($p<0.01$), suggesting degradation of cGMP in the presence of a PDE inhibitor. Adrenalin (2 mg/kg, i.p.) increased the *in vivo* and post-mortem cAMP and cGMP levels significantly ($p<0.005$, $p<0.05$, $p<0.01$, $p<0.01$, resp.). Adrenergic β-receptor blockade with propranolol (4 mg/kg, i.p.) prevented the post-mortem rise in cAMP level ($p<0.01$), while adrenergic α-receptor blockade with phenoxybenzamine (4 mg/kg, i.p.) enhanced it ($p<0.01$), but neither drug affected cGMP levels. In adrenalectomized rats, either with or without glucocorticoid substitution, the cAMP level did not change during the post-mortem period and was significantly ($p<0.02$) lower than in sham-operated controls. These data indicate that gastric mucosal cAMP formation after delayed tissue fixation is due to β-adrenoreceptor stimulation by endogenous adrenalin, and failure to employ an adequate tissue fixation technique falsifies the estimations of *in vivo* cAMP and cGMP levels by elevating cAMP and lowering cGMP concentrations, implying that various recently published studies may not accurately describe the *in vivo* condition of the rat gastric mucosa. The results indicate that MW irradiation is an efficient method for fixation of the rat gastric mucosa.

0841 EFFECTS OF ANTRAL ETHANOL ON GASTRIC ACID SECRETION, POTENTIAL DIFFERENCE, AND SERUM GASTRIN. (Eng.) Bo-Linn, G. W.; Shanbour, L. L. (Baylor Coll. Medicine, Houston, TX). *Proc. Soc. Exp. Biol. Med.* 155(4):594-598; 1977.

The effect of antral ethanol perfusion on canine gastric acid secretion and transmucosal potential difference was studied using an *in vivo*, chambered fundic segment and isolated antral pouch preparation with intact blood supply. The preparation was responsive to exogenous gastrin with pentagastrin infusion (6 μg/kg/hr) and to endogenous gastrin with acetylcholine chloride (0.5%) and glycine (1 M) perfusion of the antral pouch. The peripheral venous serum gastrin concentration increased in response to antral perfusion with glycine with a slight increase in fundic acid secretion. A small increase in peripheral serum gastrin without a corresponding increase in acid output occurred during antral perfusion with 20% ethanol. No significant differences were found in either potential difference or acid output between saline- and ethanol-treated groups, or between histamine- and histamine-plus-ethanol-treated groups. These data suggest that ethanol-stimulated antral gastrin release is insufficient to stimulate or potentiate gastric acid secretion during acute conditions in the anesthetized dog.

0842 INTRAGASTRIC TITRATION OF ACID IN THE PERFUSED RAT STOMACH PREPARATION FOLLOWING PARENTERAL AND INTRALUMINAL STIMULATION. (Eng.) Halter, F.; Galeazzi, R.; Leuenberger, J.; Smith, G. M. (Univ. Hosp., Inselspital, Berne, Switzerland). *Eur. J. Pharmacol.* 43(3):247-252; 1977.

To develop an animal model for the clinical study of gastric acid secretion, the anesthetized rat stomach preparation was adapted to assay intraluminally administered stimulants and inhibitors of acid secretion by continuously recirculating the perfusate (15 ml of 5% glucose) and measuring acid by the pH-stat technique. The responses obtained were linear. Continuous artificial neutralization

of the stomach to pH 5.5 as well as variation of the pH between 6.5 and 4.0 did not significantly alter the sensitivity of the preparation to i.v. administered human gastrin-heptadecapeptide I (HG I). Basal acid secretion, however, significantly declined following stepwise lowering of the intragastric pH between 6.5 and 4.0. The threshold values for responses to i.v. HG I were below 16 ng. Six doses could be given per rat without tachyphylaxis. Acetylcholine (1.3, 2.5, 5.2, and 10.4 µg/ml) in the perfusate induced a dose-dependent linear (r=0.902) acid secretion. Metiamide (1.66 mg/ml) in the perfusate produced a parallel shift to the right of the HG I dose-response curve, producing an agonist dose ratio of 3.66. These data suggest that intragastrically applied stimulants and inhibitors of acid secretion can be studied precisely in this relatively simple preparation.

0843 GASTRIC SECRETORY EFFECT OF AMINO ACIDS GIVEN ENTERALLY AND PARENTERALLY IN DOGS. (Eng.) Landor, J. H.; Ipapo, V. S. (CMDNJ-Rutgers Medical Sch., P.O. Box 101, Piscataway, NJ 08854). *Gastroenterology* 73(4):781-784; 1977.

To investigate the possibility that the products of protein digestion, when absorbed into the bloodstream, might stimulate gastric secretion, the possible gastric secretory effect of the parenteral administration of an amino acid (AA) mixture was determined, and this effect was compared with that produced by the enteral administration of a similar AA solution. Heidenhain pouches, gastrostomies, and Roux-en-Y jejunostomies were constructed in five mongrel dogs. A 3% solution of mixed amino acids was infused at a constant rate (120 ml/hr) throughout a 2-hr period either i.v. or intrajejunally, and the secretory response of the pouches was compared to that obtained during similar infusions of 0.9% NaCl. The i.v. administration of saline did not stimulate gastric secretion. The mean secretory responses were intrajejunal saline 37 µEq/2 hr; i.v. amino acids, 330 µEq/2 hr; and intrajejunal amino acids, 301 µEq/2 hr. These results suggest that amino acids, absorbed from the gut, may play a role in the stimulation of gastric secretion occurring when food passes through the intestine.

0844 EFFECT OF REPEATED ASPIRIN ADMINISTRATION ON THE GASTRIC MUCOSAL BARRIER AND CELL TURNOVER. (Eng.) Bolton, J. P.; Cohen, M. M. (Dept. Surgery, Univ. British Columbia, 700 West 10th Ave., Vancouver, British Columbia, V5Z IL5, Canada). *J. Surg. Res.* 23(4):251-256; 1977.

The effect of repeated aspirin administration on the canine gastric mucosa was studied by measuring gastric mucosal permeability, blood loss, and cell turnover in Heidenhain pouches. Cell turnover in the aspirin-perfused pouches (20 mM for 3 hr) was increased over that in control perfusions after a lag phase, but this did not affect the increase in gastric mucosal permeability produced by the aspirin. The increase in cell turnover may account for the observed resistance to mucosal erosions that develops after repeated aspirin administration, but the continued presence of a damaged and leaky gastric mucosal barrier may account for the failure of mucosal resistance to confer significant benefit in the form of protection from the hemorrhagic consequences of aspirin ingestion.

0845 THE EFFECT OF CIMETIDINE ON BARIUM COATING OF THE GASTRIC MUCOSA. (Eng.) James, W. B.; Stanton, B.; Teasdale, E.; McCreath, G. (Southern General Hosp., Glasgow G51 4TF, Scotland). *Br. J. Radiol.* 50(594):445-446; 1977.

The effect of cimetidine on gastric and duodenal mucosal coating by barium sulfate was investigated in 51 men referred for barium meal examination. The patients received two placebo or cimetidine tablets (400 mg) the night before the examination and two tablets (400 mg) 1 hr prior to the examination. The quality of mucosal coating in the stomach and duodenum was graded excellent, good, or poor by three observers. Although the numbers involved were too small for statistical consideration, there was judged to be no difference between the study and control groups in the quality of mucosal coating.

0846 CIMETIDINE AND THE GASTRIC MUCOSAL BARRIER. (Eng.) Kenyon, G. S.; Ansell, I. F.; Carter, D. C. (Edinburgh Univ. Medical Sch., Edinburgh, Scotland). *Gut* 18(8):631-635; 1977.

The effect of cimetidine on hydrogen and sodium transport across the gastric mucosa and its effect on taurocholate-induced permeability changes were studied in vagally-denervated pouches of antrectomized dogs. The topical or i.v. administration of cimetidine (200 mg) did not affect the net luminal Na^+ gain or net luminal H^+ loss in the pouches. The instillation of either 5 or 20 mM sodium taurocholate caused a significant increase in Na^+ net gain and H^+ net loss. The addition of 200 mg of cimetidine to either concentration of taurocholate or its administration i.v. before taurocholate administration did not reduce the net Na^+ gain or H^+ loss induced by taurocholate alone. In the case of 5 mM taurocholate, cimetidine actually increased net H^+ loss ($p<0.001$). As a potent inhibitor of gastric secretion, cimetidine could prove valuable by reducing the amount of secreted H^+ ion available to exploit a mucosal barrier damaged by agents such as bile salts.

0847 AUGMENTED GASTRIC INHIBITORY POLYPEPTIDE RESPONSE TO INTRADUODENAL GLUCOSE BY EXOGENOUS GASTRIN AND CHOLECYSTOKININ. (Eng.) Sirinek, K. R.; Cataland, S.; O'Dorisio, T. M.; Mazzaferri, E. L.; Crockett, S. E.; Pace, W. G. (Ohio State Univ. Coll. Medicine, 410 W. 10th Ave., Columbus, OH 43210). *Surgery* 82(4):438-442; 1977.

The effect of i.v. infusions of gastrin, pentagastrin, cholecystokinin, secretin, and glucagon on both fasting and glucose-stimulated gastric

inhibitory polypeptide (GIP) concentrations was studied. Five conscious dogs with Mann-Bollman fistulas each underwent six daily studies in which 50 ml of 20% glucose was infused intraduodenally (i.d.) over a period of 10 min. In five of six studies, either gastrin, pentagastrin, cholecystokinin, secretin, or glucagon was infused i.v. for 30 min before and 120 min after i.d. administered glucose. Venous blood samples at 15-min intervals were assayed for immunoreactive gastric inhibitory peptide (GIP). The i.v. administration of gastrin, pentagastrin, cholecystokinin, secretin, and glucagon had no effect on fasting immunoreactive GIP (iGIP). Compared to fasting, iGIP was significantly ($p<0.05$) increased at 15, 30, and 45 min for all treatment groups. Compared to i.d. administered glucose alone, the GIP response following either i.v. gastrin plus i.d. glucose, i.v. pentagastrin plus i.d. glucose, or i.v. cholecystokinin plus i.d. glucose was significantly greater ($p<0.05$) at 5 and 15 min. This study demonstrates augmentation of glucose-stimulated GIP by the structurally similar peptides, gastrin, pentagastrin, and cholecystokinin, suggesting a physiological role for endogenous gastrin and cholecystokinin in modulating the GIP response to glucose.

0848 ACID SECRETORY RESPONSES TO HISTAMINE, PENTAGASTRIN, AND FEEDING BEFORE AND AFTER VAGAL DENERVATION OF FUNDIC POUCHES IN CATS. (Eng.) Svensson, S. O.; Emas, S. (Karolinska Hosp., 104 01 Stockholm, Sweden). *Scand. J. Gastroenterol.* 12(3): 357-362; 1977.

The effect of local vagotomy of the fundic pouch on the acid secretory response to histamine, pentagastrin, and feeding was studied in cats. A gastric fistula (GF) of the innervated stomach, and a vagally innervated mucosal septal fundic pouch (Pavlov pouch, PP) were prepared in each of five adult cats at least 1 month prior to the experiments. Each experiment was repeated after converting the PP to a Heidenhain pouch (HP). Saline was infused i.v. (12.5 ml/hr) and histamine (10-640 µg/kg/hr) or pentagastrin (0.5-64 µg/kg/hr) was added to the infusion for 3 hr. Feeding experiments used a single meal of 3-50 g ground meat and were conducted without saline. Before denervation, the maximal response (MR) of the GF to histamine and pentagastrin was 1.04 ± 0.08 and 0.96 ± 0.05 mEq of acid/15 min, resp., and the dose-response (DR) curves levelled off at 160 and 8 µg/kg/hr for histamine and pentagastrin, resp. The D_{50} was 42 ± 9 and 2.6 ± 0.8 µg/kg/hr for histamine and pentagastrin, resp. Denervation did not affect these secretory parameters for histamine in the GF, but did elevate the MR ($p<0.01$) and lower the D_{50} ($p<0.01$) for pentagastrin. In the PP, the MR to histamine, pentagastrin, and feeding were approximately equal (0.36, 0.33, and 0.31 mEq/15 min, resp.); the DR curve plateaued at 160 and 32 µg/kg/hr of histamine and pentagastrin, resp., and at 12.5 g of meat. The D_{50} for histamine and pentagastrin was 29 ± 4 and 2.0 ± 0.7 µg/kg/hr, resp. Denervation to produce the HP markedly reduced the MR to histamine, pentagastrin, and feeding (0.20, 0.21, and 0.13 mEq/15 min, resp.); the DR curve plateau became variable for both histamine and pentagastrin and was markedly depressed for feeding; the D_{50} for histamine did not change (45 ± 6 µg/kg/hr), and was significantly ($p<0.05$) for pentagastrin. Denervation reduced the acid response to histamine by decreasing the secretory capacity, and to pentagastrin by decreasing both the secretory capacity and the sensitivity of the oxyntic glands (in this respect, the effect of local and truncal vagotomy is equal in cats). The reduction of the response to feeding was greater than to histamine or pentagastrin, which indicates that feeding does not release enough gastrin to induce the maximal rate of acid secretion in the absence of vagal impulses. But, because feeding and pentagastrin produced equal MR before denervation, it appears that vagal impulses during feeding produce greater enhancement than does basal vagal tone of the acid response to submaximal doses of gastrin.

0849 CHANGES IN PROTEIN AND NUCLEIC ACID SYNTHESIS IN RAT GASTRIC MUCOSA AFTER PENTAGASTRIN. (Eng.) Enochs, M. R.; Johnson, L. R. (Dept. Physiology, Univ. Texas Medical Sch., Houston, TX 77030). *Am. J. Physiol.* 232(2):E223-E228; 1977.

Pentagastrin was administered to rats to determine the effect of a single dose of the substance on protein and nucleic acid syntheses in the gastric mucosa. Fasted rats received i.p. either 250 µg/kg of pentagastrin or an equal volume of saline at intervals from 0 to 24 hr prior to sacrifice. Tissue from the oxyntic gland area and the duodenum were then minced and incubated in a ^{14}C-amino acid mixture (2 µCi/ml) to measure *in vitro* amino acid incorporation. Amino acid incorporation into stomach tissue and duodenal protein was significantly elevated ($p<0.05$ and 0.025, resp.) by 6 hr, and the secreted protein was significantly less ($p<0.01$) than in controls. The inhibitory effect of actinomycin D on protein synthesis was tested by comparing the incorporation of ^{14}C-amino acids into oxyntic glandular mucosa in rats that had received i.p. injections of either actinomycin D (100 µg at 24 and 7 hr before sacrifice), pentagastrin (250 µg/kg at 6 hr before sacrifice), actinomycin D and pentagastrin (schedules as given for solo doses), or saline. Pentagastrin significantly ($p<0.005$) increased incorporation of label into total gastric mucosal protein; pentagastrin plus actinomycin D significantly decreased protein synthesis compared to controls ($p<0.001$); actinomycin D alone had no significant effect. The influence of pentagastrin on nucleic acid synthesis was measured by injecting rats with either 250 µg/kg of pentagastrin or with saline at various times before sacrifice; all animals received 200 µCi ^{3}H-orotic acid i.p. 2 hr prior to sacrifice. At 1 and 2 hr after its injection, pentagastrin significantly increased incorporation of ^{3}H-orotic acid into messenger RNA (from 0.45 and 0.39 to 0.72 and 0.55 disintegrations per min [dpm]/mg DNA, resp.). Pentagastrin also induced a biphasic increase in ribosomal RNA and transfer RNA that reached a maximum of 4.59 dpm/mg

by 12 hr (control=2.82 dpm/mg). The results indicate that the main trophic effect of pentagastrin on rat gastric mucosa was an increase in protein synthesis, dependent on and preceded by increased RNA synthesis.

0850 INHIBITION OF GLUCOSAMINE SYNTHESIS BY SALICYLATES, HYDROCORTISONE AND TWO NON-ULCEROGENIC DRUGS. (Eng.) Goodman, M. J.; Kent, P. W.; Truelove, S. C. (Dept. Medicine, Univ. Chicago, Chicago, IL). *Arch. Int. Pharmacodyn.* 226(1):4-10; 1977.

Glucosamine synthesis in homogenates of human gastric and colonic mucosa was studied in the presence of the ulcerogenic drugs sodium salicylate, acetylsalicylic acid, and hydrocortisone hemisuccinate, and in the presence of the nonulcerogenic analgesic, paracetamol. The nonulcerogenic antibiotic, gentamicin, was tested for its effect upon glucosamine synthesis in a homogenate of human colonic mucosa. The homogenates (50 mg tissue/ml) were incubated in 1.0 ml volumes of substrate buffer mixture containing 1, 2, 5, or 10 mg of a drug (only 1, 2, or 5 mg for acetylsalicylic acid) 37 C for 3 hr. Concentrated perchloric acid (71%) was used to stop the reaction. After centrifugation, glucosamine was assayed by the Morgan-Elson color reaction. Dose-related inhibition of glucosamine synthesis was observed with each drug. Sodium salicylate, acetylsalicylic acid, and paracetamol produced 50% inhibition at concentrations of 27, 23, and 29 mM, resp., while hydrocortisone hemisuccinate produced 50% inhibition at 10 mM. Gentamicin inhibited glucosamine synthesis to approximately the same extent as the salicylates. The results indicate that the ulcerogenic actions of the salicylates and hydrocortisone hemisuccinate are not due to the inhibition of glucosamine synthesis in the mucosa.

0851 INHIBITION BY SOMATOSTATIN OF GASTRIN RELEASE AND GASTRIC ACID RESPONSES TO MEALS AND TO PENTAGASTRIN IN MAN. (Eng.) Philip, J.; Domschke, S.; Domschke, W.; Urbach, H. J.; Reiss, M.; Demling, L. (Dept. Medicine, Univ. Erlangen-Nurnberg, Erlangen, W. Germany). *Scand. J. Gastroenterol.* 12(3):261-265; 1977.

Somatostatin was administered i.v. to six healthy male volunteers to investigate its effect on meal- and pentagastrin-induced acid secretion and gastrin release. Somatostatin (5 µg/kg/hr, cyclic form) was administered during the second hour of a 3-hr period of stimulation with either pentagastrin (1.5 µg/kg/hr, i.v.) or a test meal (10% peptone, pH 5.5). A modified Fordtran and Walsh method of intragastric titration was used to determine meal-induced gastric acid secretion. Somatostatin decreased the pentagastrin-induced acid response achieved by the first hour of stimulation by 68 ± 17% ($p<0.001$) and by 87 ± 2% ($p<0.001$) during the last 30 min of somatostatin infusion. The total 1-hr pepsin output was decreased by 72 ± 14% ($p<0.001$) and by 84 ± 2% ($p<0.001$) during the last 30 min. The total 1-hr meal-induced acid secretion was reduced by 75 ± 21% ($p<0.001$) by somatostatin (by 96 ± 3% [$p<0.001$] during the last 30 min of infusion). Meal-stimulated gastrin levels, nearly twice the fasting levels, were decreased only 27 ± 9% ($p<0.02$) even during the second 30 min of infusion. The results suggest that somatostatin inhibits meal-induced acid secretion mainly by a direct effect on the parietal cells and only partially by indirect inhibition via suppression of gastrin release.

0852 EFFECT OF SOMATOSTATIN ON GASTROINTESTINAL SECRETIONS AND PEPTIC ULCER PRODUCTION IN CATS. (Eng.) Konturek, S. J.; Radecki, T.; Pucher, A.; Coy, D. H.; Schally, A. V. (Inst. Physiology, Medical Acad., Krakow, Poland). *Scand. J. Gastroenterol.* 12(3):379-383; 1977.

Somatostatin (GH-RIH) was infused i.v. into conscious cats to study its effect on gastric and pancreatic secretion and the formation of peptic ulcers. Chronic gastric and pancreatic fistulas were prepared in six cats, and a gastric (pentagastrin or histamine, i.v.) or pancreatic (secretin, i.v. or intraduodenal acid) stimulant was administered to produce maximal secretion. After 90 min of secretory stimulation, GH-RIH (0.62-5.0 µg/kg/hr) was infused i.v. for 1 hr, followed by an additional 60-min administration of the stimulant. Peptic ulcer formation was studied in 48 cats after a 36-hr perfusion with one of the following solutions: histamine (160 µg/kg/hr) or pentagastrin (8 µg/kg/hr) alone or with GH-RIH (2 µg/kg/hr). GH-RIH produced a dose-related inhibition of pentagastrin-stimulated acid secretion, reaching approximately 50% inhibition at a dose of 2 µg/kg/hr. Pepsin inhibition was about 90% at 1 µg/kg/hr of GH-RIH, but was not dose-related. GH-RIH had no effect on histamine-stimulated acid output, but did significantly decrease pepsin output. GH-RIH (2 µg/kg/hr) almost completely inhibited secretin-stimulated pancreatic bicarbonate secretion, and strongly inhibited bicarbonate secretion stimulated by intraduodenal acid, but did not affect protein secretion induced by either stimulant. GH-RIH significantly reduced the incidence and size of duodenal ulcers induced by pentagastrin. Peptic ulcers and related complications were also decreased by GH-RIH. It is concluded that GH-RIH may directly affect the oxyntic glands, but not the histamine receptors. Furthermore, GH-RIH may be useful in treatment of peptic ulcers in man.

0853 EFFECT OF SOMATOSTATIN ANALOGS ON GASTRIC AND PANCREATIC SECRETION. (Eng.) Konturek, S. J.; Tasler, J.; Krol, R.; Dembinski, A.; Coy, D. H.; Schally, A. V. (Medical Acad., Krakow, Poland). *Proc. Soc. Exp. Biol. Med.* 155(4):519-522; 1977.

Somatostatin and its two analogs, D-^{8}Trp-somatostatin and D-^{14}Cys-somatostatin were tested for their effects on gastric and pancreatic secretion in chronic gastric and pancreatic fistula dogs. All three peptides, infused i.v. at 2.5 µg/kg/hr, caused simi-

lar competitive inhibition of pentagastrin-induced gastric acid and secretin-induced pancreatic bicarbonate secretions. During somatostatin infusion, the dose-response curve for acid secretion from the stomach was shifted to the right; there was an almost threefold increase in K_m, while V_{max} was only slightly decreased. The infusion of either analog produced a similar shift of the dose-response curve, a doubling of K_m, and a small reduction in V_{max}. Somatostatin and its analogs all shifted the dose-response curve for pancreatic bicarbonate secretion to the right, increased the K_m, and slightly reduced V_{max}. The results indicate that somatostatin analogs, which were shown to have a dissociated action on pituitary and endocrine pancreatic secretion, are essentially as equipotent as somatostatin itself on gastric and pancreatic secretions.

0854 ANTI-STRESS ULCER AND ANTI-SECRETORY EFFECT OF SOMATOSTATIN IN RATS--FAILURE TO SUPPRESS SERUM GASTRIN. (Eng.) Schwille, P. O.; Putz, F.; Thun, R.; Schellerer, W.; Draxler, G.; Lang, G. (Chirurgische Universitatsklinik, Maximiliansplatz D-8520 Erlangen, W. Germany). *Acta Hepato-gastroenterol.* 24(4):259-265; 1977.

The effect of somatostatin on stress ulceration and on gastrin secretion was investigated in rats. Some rats were restrained so that free movement was restricted, while others were completely restrained. Complete restraint reduced the volume of gastric secretion, acid and pepsin secretion, the acid concentration, and aminopyrine clearance, while the ulcer index was elevated. During both types of restraint, somatostatin inhibited the volume and acid secretion in a dose-dependent manner and lowered the ulcer index. During continuous somatostatin infusion (10.0 µg/kg/hr, 8 hr), the acid concentration and acid and pepsin secretion fell. The ulcer index of gastric fistula in somatostatin-treated rats dropped to 50% of that in vehicle-treated controls. Microcirculation and serum gastrin levels were unchanged. In animals without gastric fistula, the ulcer index was reduced by 85%; the serum gastrin level was unchanged. It is concluded that stress ulcers develop at a low secretory and microcirculatory state of gastric mucosa. The prophylactic effect of somatostatin results from direct inhibition of parietal and chief cells.

0855 ACTION OF PROGLUMIDE ON GASTRIC ACID SECRETION AND MOTILITY STIMULATED BY PENTAGASTRIN IN THE NONANESTHETIZED CAT. (Fre.) Desvigne, C.; Vagne, M.; Faivre, M.; Desvigne, A. (Hopital E.-Herriot, Pavillon H, F 69374 Lyon Cedex 2, France). *Gastroenterol. Clin. Biol.* 1(5):447-454; 1977.

0856 CHOLESTEROL BIOSYNTHESIS IN STOMACH AND INTESTINE TISSUES OF RATS. (Ukr.) Nikolenko, I. A.; Perederij, O. F. (A. V. Palladin Inst. Biochemistry, Acad. Sciences, Kiev, Ukrainian SSR). *Ukr. Biokhim. Zh.* 49(5):70-75; 1977.

0857 GASTRIC ANTISECRETORY ACTIONS OF (15s) 15 METHYL PROSTAGLANDIN E_2 METHYL ESTER AND NATURAL PROSTAGLANDIN E_2 IN RHESUS MONKEYS. (Eng.) Dajani, E. Z.; Callison, D. A. (Searle Lab., Chicago, IL 60680). *Prostaglandins* 11(5):799-808; 1976.

0858 GASTROSCOPIC OBSERVATION OF THE EFFECT OF VARIOUS ANTIINFLAMMATORY DRUGS ON THE GASTRIC MUCOSA [Abstract]. (Eng.) Hradsky, M.; Bruce, L. (Falu Hosp., Falun, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):35; 1977.

0859 EFFECT OF IONIC STRENGTH ON THE PROTEOLYTIC CHARACTERISTICS OF HUMAN PEPSINS [Abstract]. (Eng.) Samloff, I. M.; Dadufalza, V. (Harbor General Hosp., Los Angeles, CA). *Gut* 18(11):A949-A950; 1977.

0860 ROLE OF CYCLIC 3',5'-AMP-DEPENDENT PHOSPHORYLATION OF MEMBRANE PROTEINS IN THE ACTIVATION OF OXYNTIC CELLS. (Eng.) Salganik, R. I. (Inst. Cytology, Genetics, Siberian Branch of the USSR Acad. Sciences, Novosibirsk, USSR). *Gastroenterology* 73(4, Part 2):920; 1977.

0861 INHIBITION OF BILE SALT-INDUCED GASTRIC MUCOSAL EROSIONS BY 16,16-DIMETHYL PROSTAGLANDIN E_2 IN THE RAT [Abstract]. (Eng.) Main, I. H. M.; Melarange, R. (Sch. Pharmacy, Univ. London, 29/39 Brunswick Square, London WC1N 1AX, England). *Br. J. Pharmacol.* 61(1): 139P; 1977.

0862 EXPERIMENTAL GASTRIC ULCER BY INJECTION OF ACETIC ACID INTO THE GASTRIC WALL. (Jpn.) Oku, K. (Wakayama Medical Coll., Wakayama, Japan). *J. Wakayama Med. Soc.* 27(3):133-147; 1976.

0863 DUODENAL INHIBITION OF GASTRIN RELEASE IN DOGS [Abstract]. (Eng.) Rokkjaer, M.; Host, V.; Brandsborg, O. (Arhus Kommunehospital, Arhus, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):80; 1977.

0864 CHANGES IN GASTRIC MORPHOLOGY AND FUNCTIONS AFTER CORTICOSTEROID ADMINISTRATION IN DOGS [Abstract]. (Eng.) Holter, O.; Stave, R. (Natl. Hosp. Norway, Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):34; 1977.

0865 PLASMA GASTRIN AND GASTRIC SECRETORY RESPONSE TO DUODENAL PERFUSION WITH LIVER EXTRACT IN HEALTHY HUMAN SUBJECTS [Abstract]. (Eng.) Grabner, P.; Semb, L. S.; Schrumpf, E.; Myren, J.; (Diakonhjemmets Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):26; 1977.

0866 THE INHIBITORY EFFECT OF A SYMPATIC STIMULANT ON THE ACID SECRETION IN THE DOG [Abstract]. (Eng.) Gottrup, F.; Ornsholt, J.; Andersen, D. (Univ. Dept. Surgical Gastroenterology, Amtsygehuset, Aarhus, Denmark). *Gastroenterology* 12(Suppl. 45):25; 1977.

0867 EFFECT OF DOXEPIN ON GASTRIC SECRETION IN MAN [Abstract]. (Eng.) Giercksky, K. E.; Oyen, D.; Burhol, P. G.; Johnson, J. A. (Surgical Dept., Univ. Tromso, Tromso, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):23; 1977.

0868 FAT INDUCED JEJUNAL INHIBITION OF MEAL STIMULATED GASTRIC ACID SECRETION AND RELEASE OF GIP, GLUCAGON, GLI, AND VIP IN HEALTHY HUMAN SUBJECTS [Abstract]. (Eng.) Bech, A.; Christiansen, J.; Lauridsen, K.; Holst, J. J.; Fahrenkrug, J.; Schaffalitzky de Muckadell, O. (Glostrup Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):7; 1977.

0869 EFFECTS OF INTRAVENOUS CIMETIDINE ON GASTRIC SECRETION IN RESPONSE TO INTRAVENOUS HISTAMINE AND PENTAGASTRIN IN MAN [Abstract]. (Eng.) Aadland, E.; Berstad, A. (Aker Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45): 117; 1977.

0870 EFFECT OF SYMPATHIC NERVE BLOCKADE ON GASTRIC ACID SECRETION IN GUINEA PIGS [Abstract]. (Eng.) Aaraas, I.; Gauperaa, T. (Dept. Anaesthesiology, Univ. Tromso, Tromso, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45): 118; 1977.

0871 BETHANECHOL AND ATROPINE BOTH INHIBIT THE GASTRIN RESPONSE TO BOMBESIN [Abstract]. (Eng.) Taylor, I. L.; Walsh, J. H.; Carter, D. C.; Chew, P.; Grossman, M. I. (Veterans Admin. Wadsworth Hosp. Center, Los Angeles, CA). *Clin. Res.* 25(4): 574A; 1977.

0872 BOMBESIN IS A POTENT STIMULANT OF PANCREATIC POLYPEPTIDE (PP) RELEASE [Abstract]. (Eng.) Taylor, I. L.; Walsh, J. H.; Wood, J.; Chew, P.; Carter, D. (Veterans Admin. Wadsworth Hosp. Center, Los Angeles, CA). *Clin. Res.* 25(4):574A; 1977.

0873 INHIBITION OF GUINEA PIG FUNDIC GASTRIC MUCOSA ADENYLATE CYCLASE (AC) BY PROSTAGLANDIN PRECURSORS [Abstract]. (Eng.) Dozois, R. R.; Dousa, T. P. (Mayo Clinic and Foundation, Rochester, MN). *Clin. Res.* 25(4):607A; 1977.

0874 EFFECT OF PARENTERAL ASPIRIN ON THE HISTOLOGY AND POTENTIAL DIFFERENCE OF HUMAN GASTRIC MUCOSA [Abstract]. (Eng.) Paone, D. B.; Krause, W. J.; Ivey, K. J. (Harry S Truman Veterans Admin. Hosp., Columbia, MO). *Clin. Res.* 25(4):608A; 1977.

0875 EFFECT OF MANNITOL ON RAT GASTRIC MUCOSAL POTENTIAL DIFFERENCE [Abstract]. (Eng.) Enochs, M. R.; Ivey, K. J.; Burks, M.; Ackman, K. *Clin. Res.* 25(4):571A; 1977.

0876 COMPARATIVE EFFECTS OF FENOPROFEN, KETOPROFEN, IBUPROFEN AND NAPROXEN ON GASTRIC MUCOSA [Abstract]. (Eng.) Mann, N. S.; Mann, S. K.; Borkar, B. B. (Veterans Admin. Hosp., Louisville, KY). *Clin. Res.* 25(4):572A; 1977.

0877 COMPARATIVE EFFECTS OF ASPIRIN AND FENOPROFEN ON GASTRIC MUCOSA [Abstract]. (Eng.) Mann, N. S.; Mann, S. K.; Borkar, B. B. (Veterans Admin. Hosp., Louisville, KY). *Clin. Res.* 25(4):572A; 1977.

See also, 0783, 0821, 0835, 1019, 1051, 1054, 1057, 1058, 1060, 1062, 1067, 1068, 1079, 1207, 1209, 1343.

SECRETION AND METABOLISM

Pancreas

0878 PURIFICATION OF PLASMA MEMBRANES FROM RAT PANCREAS: A RAPID METHOD. (Eng.) Poirier, G. G.; Lambert, M. P.; Lebel, D.; Sakr, F.; Morisset, J.; Beaudoin, A. R. (Faculty of Sciences, Univ. Sherbrooke, Quebec J1K 2R1, Canada). *Proc. Soc. Exp. Biol. Med.* 155(3):324-329; 1977.

A rapid and simple method for the isolation of plasma membranes from the rat pancreas is described. Pancreases are homogenized in buffer A. The homogenate is filtered through cheesecloth and centrifuged at 50 x *g* for 5 min to remove RBC, unbroken cells, and connective tissue. The supernatant is then centrifuged at 5,100 x *g* for 20 min. The top layer of the 5,100 x *g* pellet is removed for further purification on a sucrose gradient. The separated layer is mixed with 75% sucrose to a final concentration of 40% and inserted in a

stepwise sucrose gradient of 25, 30, 35, and 40% (containing the 5,100 x *g* pellet). This gradient is centrifuged for 1 hr at 115,000 x *g*. The different fractions are removed with a syringe and diluted with buffer B containing 40 mM Tris-HCl, pH 7.4, 0.25 M sucrose, 1 mg/ml albumin, and 5 mM β-mercaptoethanol. They are then centrifuged at 30,000 x *g*. Electron microscopy of the material recovered at the interface of sucrose 30-35% showed that it consisted mainly of smooth vesicular membranes, although some contamination by rough endoplasmic reticulum was observed. The specific activity of adenylate cyclase in the material sedimenting at the interface 30-35% and identified as smooth vesicular membranes by electron microscopy demonstrated a 44-fold increase in activity in comparison with that in the homogenate. The succinate dehydrogenase activity, a mitochondrial marker, was low in the adenylate-cyclase-rich fraction, and α-amylase was practically absent from the purified fraction. There was a sevenfold decrease in RNA content as compared to the total homogenate, which could be explained by the small contamination by rough endoplasmic reticulum. The optimal concentration of secretin necessary to obtain maximal stimulation of adenylate cyclase activity was 2 clinical U/ml. This method has several improvements over other techniques: the new finding that succinate dehydrogenase activity is decreased and the use of the RNA content as a marker for rough endoplasmic reticulum contamination. This method also provides an excellent tool for the study of the interaction of secretin and other hormones with the membrane.

0879 EXOCRINE PANCREAS RESPONSE TO A TEST MEAL IN THE DOG: CHANGES INDUCED BY 3 MONTHS' ETHANOL FEEDING. (Eng.) Tiscornia, O. M.; Singer, M.; Mendes De Oliveira, J. P.; Demol, P.; Sarles, H. (Unite de Recherches de Pathologie Digestive, INSERM U31, 46 chemin de la Gaye, 13009 Marseilles, France). *Am. J. Dig. Dis.* 22(9):769-774; 1977.

Pancreatic secretion in response to a test meal was compared in nonalcoholic dogs and in those fed ethanol for 3 months. Three dogs were equipped with a chronic gastric fistula (Thomas cannula) and a new type of chronic pancreatic fistula that allows collection of pure nonactivated pancreatic juice after ingestion of a test meal. During the nonalcoholic period, a test meal (400 g canned dog meal) was given simultaneous with an infusion of 200 ml isotonic saline through the gastric cannula. Volume, bicarbonate concentration and output, and protein output showed a peak 40 min after ingestion of the test meal. Protein concentration showed an opposite pattern. During the alcoholic period, the animals were given 50% ethanol (2.0 g/kg/day) associated with a protein-supplemented diet through the gastric cannula, and test meal experiments were repeated at the end of this period. When compared with the nonalcoholic period, the secretory response to the meal was significantly modified; volume (-42%) and bicarbonate concentration (-19%) and output (-54%) were significantly lower, protein output remained unchanged, and protein concentration increased (+74%). All three dogs excreted protein plugs through the catheter collecting pancreatic juice from the main pancreatic duct. It is postulated that a depression of secretin release and an increased gastrin release and cholinergic tone could be the basis of the ethanol-evoked change in response to a test meal.

0880 CANINE PANCREATIC EXOCRINE SECRETORY RESPONSE TO LOW- AND HIGH-FAT ELEMENTAL DIETS. (Eng.) Lewis, J. W.; Freeman, J. B. (Univ. Iowa Hosp. and Clinics, Iowa City, IA 52242). *Can. J. Surg.* 20(4):345-348; 1977.

The response of the dog exocrine pancreas to intragastric infusions of a commercially available diet with 30% of the calories present as fat (Flexical) was compared to that of a similar diet that was essentially fat-free (Vivonex). Each of these diets, as well as a standard tube feeding formula, was infused at the rate of 2 ml/min for 2 hr on each of 3 days. The volume and protein and bicarbonate content of eight 15-min samples of pancreatic secretion collected via a pancreatic pouch in the 2-hr period were measured. The high-fat elemental diet produced significantly ($p<0.05$) greater volume and bicarbonate output than did the low-fat elemental diet or the standard formula. Protein content of the pancreatic secretion was significantly ($p<0.01$) reduced during infusions of either low- or high-fat elemental diets compared to the standard diet, indicating decreased enzyme output. It is concluded that low-fat elemental diets are preferable for enteral nutrition in patients with pancreatic disorders where limitation of pancreatic secretions is desirable.

0881 CALCITONIN AND EXOCRINE PANCREATIC SECRETION IN MAN: INHIBITION OF ENZYMES STIMULATED BY CCK-PANCREOZYMIN, CAERULEIN, OR CALCIUM--NO RESPONSE TO VAGAL STIMULATION. (Eng.) Hotz, J.; Goebell, H.; Ziegler, R. (Medical Clinic, Univ. Essen, Hufelandstr. 55, D4300 Essen 1, W. Germany). *Gut* 18(8):615-622; 1977.

The effect of calcitonin on human pancreatic secretion was studied under various conditions of stimulation. During a continuous i.v. infusion of both secretin plus cholecystokinin-pancreozymin (CCK-PZ, 1 U/kg/hr for each hormone) or secretin plus cerulein (1 U/kg/hr secretin, 75 μg/kg/hr cerulein), enzyme secretion was promptly reduced by an infusion of calcitonin (60 Medical Research Council U/hr) by more than 50%. In contrast, vagally stimulated enzyme secretion induced by insulin-hypoglycemia or carbamyl-choline was not altered against a background infusion of calcitonin in comparison with control experiments. Calcium-induced enzyme secretion was abolished by additional calcitonin infusion, which also prevented an increase in serum calcium. An additional high grade calcium infusion did not modify the inhibitory action of calcitonin on enzyme output stimulated by secretin and CCK-PZ. Fluid and bicarbonate secretion remained unaffected by calcitonin in all ex-

perimental conditions, whereas the outputs of calcium and magnesium paralleled generally the changes in enzymes. It is suggested that calcitonin interferes with hormone-mediated stimulation of the acinar cells without influencing cholinergic mechanisms. The inhibitory action of calcitonin on enzyme secretion does not appear to be mediated by a depletion of extracellular calcium in the pancreatic tissue by calcitonin.

0882 IN VITRO EFFECTS OF SECRETIN AND VASOACTIVE INTESTINAL POLYPEPTIDE ON HYDROLASE SECRETION AND CYCLIC AMP LEVELS IN THE PANCREAS OF FIVE ANIMAL SPECIES. A COMPARISON WITH CERULEIN. (Eng.) Robberecht, P.; Deschodt-Lanckman, M.; Lammens, M.; De Neef, P.; Christophe, J. (Faculte de Medecine, Universite libre de Bruxelles, bvd de Waterloo 115, B-1000 Brussels, Belgium). *Gastroenterol. Clin. Biol.* 1(6/7):519-525; 1977.

The *in vitro* effects of secretin, vasoactive intestinal polypeptide (VIP), and cerulein on hydrolase (amylase and lipase) secretion in pancreas fragments from the rat, mouse, guinea pig, cat, and dog were studied. Cyclic AMP levels were also measured to determine a correlation with hydrolase secretion. Cerulein stimulated pancreatic hydrolase secretion in all five species. Secretin and VIP were potent stimuli of enzyme secretion in the rat and guinea pig only. A greater amylase response to VIP than to secretin was observed in the rat, whereas the opposite was true in the guinea pig. The peptide concentration required for maximal stimulation was 100-fold higher for secretin or VIP than for cerulein. Cyclic AMP levels increased in pancreas fragments from all five species exposed to secretin or VIP, but cerulein had no effect on the intracellular levels of cyclic AMP. No quantitative correlation between cyclic AMP increase and amylase secretion in response to VIP and secretin was observed in the rat; in the guinea pig, VIP was a better stimulus than secretin of hydrolase secretion, and the reverse was true for the cyclic AMP response. These data confirm that in the rat and guinea pig pancreatic hydrolase secretion is stimulated *in vitro* by two different mechanisms, one involving cyclic AMP and the other involving cyclic guanosine 3',5' monophosphate and calcium.

0883 BIOCHEMICAL REACTIONS INVOLVED IN PANCREATIC ENZYME SECRETION. 2. INHIBITORY EFFECTS OF TETRACAINE AND ATROPINE. (Eng.) Morisset, J.; Beaudoin, A. R. (Dept. Biology, Univ. Sherbrooke, Sherbrooke, Quebec J1K 2R1, Canada). *Can J. Physiol. Pharmacol.* 55(3):639-643; 1977.

The inhibitory effects of tetracaine on cholecystokinin-pancreozymin (CCK-PZ)- and urecholine-stimulated pancreatic enzyme secretion and the potentiating effect of these drugs on dibutyryl cyclic AMP-stimulated enzyme secretion were investigated in male Sprague-Dawley rats; the effect of tetracaine on calcium efflux was also studied. Tetracaine pretreatment (2 mM, 30 min) did not modify basal amylase release, but the secretory response to urecholine (10^{-5} M) was subsequently reduced by 70%. The response to CCK-PZ (2 Ivy U) was not affected. When tetracaine was added to medium already containing both CCK-PZ and urecholine (10 min after the start of incubation), amylase release was not significantly affected. Co-addition of atropine (10^{-5} M) and urecholine to pancreatic tissue for 30 min resulted in complete inhibition of amylase secretion. The addition of atropine to tissue already stimulated by the cholinergic drug was associated with a 48% decrease in amylase secretion. The secretory response to CCK-PZ was not modified by atropine. Both CCK-PZ and tetracaine increased calcium efflux over the control level when added separately; when combined, a further increase in calcium release was observed. Urecholine caused a rapid release of calcium within the first 15 min, but addition of tetracaine to the cholinergic was not associated with a further increase in calcium efflux. When dibutyryl cyclic AMP was substituted for CCK-PZ and urecholine, there was a significant increase in calcium efflux that was less pronounced than that after CCK-PZ or urecholine. The effect of tetracaine on calcium efflux might partly explain the inhibitory action of the drug.

0884 EXOCRINE PANCREATIC INSUFFICIENCY SYNDROME IN CBA/J MICE. III. PATHOLOGICAL AND GENETIC ANALYSIS. (Eng.) Leiter, E. H.; Cunliffe-Beamer, T. (Jackson Lab., Bar Harbor, ME). *Gastroenterology* 73(2):260-266; 1977.

The clinical and histopathological characteristics of exocrine pancreatic insufficiency (EPI) syndrome were studied in CBA/J mice fed diets of protein and fat *ad libitum*. Exocrine pancreatic function was measured by a gelatin digestion test for fecal proteolytic activity. Two groups of 7- to 11-week-old female EPI mice were given protein/fat diets supplemented with either 0.5 or 1.0 g of pancreatin/30.0 g diet for 14 weeks; and weight changes during the next 17 weeks were compared with those in control mice. Plasma glucose and insulin levels were measured in EPI and normal mice and genetic analysis of the EPI syndrome was also performed. The sera of EPI mice were tested for antibody to Coxsackie viruses B1 and B6; the sera of normal CBA/J mice were tested for antibody to reovirus III; and sonicated pancreatic extracts were examined for type C group-specific viral antigen p30 by complement fixation testing. Gross and histopathological characteristics of EPI mice were emaciation, distention of the bowels, and replacement of exocrine pancreatic tissues by fat. Subclinical signs of the disease were focal areas of degeneration and inflammatory cell infiltration in the exocrine pancreas. EPI mice not treated with pancreatin had no fecal tryptic activity, but subclinical stages of the disease could not be detected by this test. Pancreatin produced significant, but reversible, increases in general health and body weight in EPI mice. EPI mice receiving long-term pancreatin treatment were normoglycemic (121 mg plasma glucose/100 ml), while untreated EPI mice were hypoglycemic (81 mg/100 ml). Both treated

and untreated mice showed normal plasma insulin levels (40-60 μU/ml), indicating that EPI syndrome did not affect islet function. Matings of normal parents, normal parents with at least one previous EPI offspring, and EPI parents produced EPI progeny at frequencies of 1.4, 2.6, and 3.8%, resp., suggesting that the disease occurs spontaneously. Preweaning mortality in mice from EPI breeders was not due to early expression of a recessive gene as the incidence of mortality (37.8%) was markedly decreased to 3.2% by fostering 1-day-old neonates to asymptomatic CBA/J females. The trait could not be outcrossed to other strains, and no evidence of viral etiology was observed. The results indicate that the cause of exocrine cell necrosis characteristic of EPI is not genetic, but that there is a genetically predisposing background in EPI in the CBA/J strain.

0885 ACUTE PANCREATITIS FOLLOWING INTRAPANCREATIC COMPLEMENT ACTIVATION IN THE RAT. (Ger.) Seelig, R.; Seelig, H. P.; Rohnacher, R. (Institut fur Immunologie und Serologie, Universitat Heidelberg, D-6900 Heidelberg, W. Germany). *Z. Gastroenterol.* 14(6):654-667; 1976.

Adult Sprague-Dawley rats were sensitized with rabbit IgG (9 mg/week for 4 consecutive weeks, s.c.), and intrapancreatic Arthus phenomenon was induced in the animals with an antibody titer of 1/8 by the intraductal injection of rabbit IgG (4 mg) 7 days after the last sensitization. The intrapancreatic immune complex reaction caused acute pancreatitis (acinus cell necrosis, intravasal leukocytosis, leukodiapedesis, leukocyte infiltration of the walls of veins and capillaries, and, ultimately, interstitial leukocyte infiltration). Rabbit IgG administered to nonsensitized rats and bovine albumin administered intraductally to sensitized rats failed to induce acute pancreatitis. Complement reactivation with acute pancreatitis similar to that seen after the intraductal administration of rabbit IgG was induced in sensitized rats by the intraductal administration of inulin (15 and 30 mg) and cobra venom factor (45 or 200 U). The localization of the early acinus cell necrosis and of the maximal leukocyte infiltration was determined basically by the site of the primary complement activation. The extent of the necrosis was dependent on the diffusion of the complement-activating substance in the interstitial edema. Following the intraductal injection of inulin and cobra venum factor, the activation of the complement system was triggered via the C3 shunt, leading the release of cytolytically active complement and biologically active mediators from the complement system, independent of immune reactions. The findings indicate that intrahepatic complement activation by immunological and non-immunological processes leads to acute necrotizing pancreatitis, with the release of cytolytically active complement being the common pathogenetic occurrence in the initial acinus cell membrane lesion.

0886 EFFECTS OF PANCREATIC DUCT LIGATURE IN RABBITS FED A TRADITIONAL DIET. (Fre.) Catala, J. (Institut de Physiologie, 2, rue Francois-Magendie, 31400 Toulouse, France). *Ann. Biol. Anim. Biochim. Biophys.* 17(4):573-578; 1977.

0887 LONG-TERM EFFECTS OF PANCREATIC DUCT LIGATURE ON DIGESTIBILITY IN THE PIG. (Fre.) Corring, T.; Bourdon, D. (Laboratoire de Physiologie de la Nutrition, I.N.R.A., 78350 Jouy-en-Josas, France). *Ann. Biol. Biochim. Biophys.* 17(4):579-582; 1977.

0888 SHORT- AND MEAN-TERM EFFECT OF PANCREATIC DUCT LIGATURE ON DIET DIGESTIBILITY IN THE FOOD-RESTRICTED GROWING RABBIT. (Fre.) Corring, T.; Lebas, F. (Laboratoire de Physiologie de la Nutrition, I.R.A., 78350 Jouy-en-Josas, France). *Ann. Biol. Anim. Biochim. Biophys.* 17(3A):299-307; 1977.

0889 EFFECT OF ALCOHOLIC INTOXICATION ON THE FUNCTION AND OXIDATIVE PROCESSES OF PANCREATIC TISSUE. (Rus.) Shishkov, V. I. (Central Scientific Res. Lab., Sverdlovsk Medical Inst., Sverdlovsk, USSR). *Farmakol. Toksikol.* 40(5):613-616; 1977.

0890 ANALYSIS OF SUBSTANCE TRANSPORT INTO ACINAR CELLS. (Rus.) Ter-Akopova, I. R.; Bukhvalov, I. B. (N. I. Pirogov Second Moscow Medical Inst., Moscow, USSR). *Biull. Eksp. Biol. Med.* 34(8):242-244; 1977.

0891 DISTRIBUTION OF ^{99m}Tc-1-THIOGLUCOSE IN RATS: EFFECT OF ADMINISTRATION ROUTE ON PANCREATIC SPECIFICITY. (Eng.) Risch, V. R.; Honda, T.; Heindel, N. D.; Emrich, J. L.; Brady, L. W. (Hahnemann Medical Coll., Philadelphia, PA 19102). *Radiology* 124(3):837-838; 1977.

See also, 0787, 0807, 0834, 0853, 1024, 1032, 1046, 1047, 1048, 1049, 1055, 1056, 1094, 1256, 1347.

SECRETION AND METABOLISM

Hepatobiliary Tract

0892 ADRENERGIC MECHANISMS IN THE HEPATIC MICROCIRCULATION IN THE RAT. (Eng.) Koo, A.; Liang, I. Y. S.; Cheng, K. K. (Dept. Physiology, Univ. Hong Kong, Sassoon Rd., Hong Kong). *Q. J.*

Exp. Physiol. 62(3):199-208; 1977.

To elucidate the role of adrenergic constrictor-dilator mechanisms in the hepatic sinusoids in the control of blood flow in the terminal hepatic microcirculation, the *in vivo* rat liver microvascular bed was examined microscopically, and the diameter of the hepatic sinusoids was measured by serial photomicrography. The intraportal infusion of epinephrine, norepinephrine, and tyramine produced a constriction of the sinusoids at all concentrations (10^{-9}-10^{-4} M), but dilatation of some sinusoids occurred when the concentrations were low (10^{-9}-10^{-7} M). The effects of tyramine on the sinusoids were attributed to the release of endogenous norepinephrine, which activated either alpha- or beta-adrenergic receptors, causing either constriction or dilatation, resp. Alpha-adrenergic blockade with phenoxybenzamine (1 mg/kg) caused a significant increase in the mean diameter of the sinusoids (6.3 ± 0.05 versus 6.5 ± 0.05 μm, $p<0.001$) after 30 min. Beta-adrenergic blockade by propranolol (1 mg/kg) induced a significant constriction of the sinusoids. All the above responses were abolished by pretreatment with reserpine. The automatic control of sinusoidal blood flow may be regulated by norepinephrine-mediated basal vasomotor tone in the hepatic sinusoids.

0893 HEPATIC SUPPORT WITH HEPATOCYTE SUSPENSIONS IN A PERMEABLE MEMBRANE DIALYZER. (Eng.) Olumide, F.; Eliashiv, A.; Kralios, N.; Norton, L.; Eiseman, B. (Denver General Hosp., Denver, CO 80220). *Surgery* 82(5):599-606; 1977.

A new concept of hepatic support with circulating hepatocytes on one side of a cuprophan membrane and blood from an anhepatic pig on the other was evaluated in 19 dialysis experiments. Levels of pyruvate metabolism (50% of an initial value of 480 counts/min in $^{14}CO_2$ produced from 2-^{14}C-pyruvate/10^6 cells after 2-4 hr of perfusion) and oxygen utilization (0.01 ml O_2/g liver/min after 4 hr of perfusion) were similar to those obtained in previous experiments, when liver cells in a centrifuge are perfused with oxygenated plasma. Of six anhepatic pigs given late hepatic support, five showed neurologic improvement. One had concomitant improvement in electroencephalographic results. A membrane hemodialysis unit in which hepatocytes circulate against blood from an anhepatic animal is the most suitable extracorporeal liver support unit described so far. Its simplicity allows repeated and prolonged use.

0894 CHANGES IN THE BLOOD-BRAIN BARRIER IN HEPATIC COMA AFTER HEPATECTOMY IN THE RAT. (Eng.) Livingstone, A. S.; Potvin, M.; Goresky, C. A.; Finlayson, M. H.; Hinchey, E. J. (Montreal General Hosp., 1650 Cedar Ave., Montreal, Quebec H3G 1A4, Canada). *Gastroenterology* 73(4): 697-704; 1977.

The possibility that the hepatic coma seen after massive liver necrosis or hepatectomy may result from an alteration in the blood-brain barrier was examined. Bloodless two-stage hepatectomies were performed on Wistar rats, and the animals were maintained with i.v. glucose infusions. When the animals were in late stage 3 or stage 4 hepatic coma, the functional integrity of the blood-brain barrier was assessed by means of the sudden arterial injection-15-sec decapitation-Oldendorf tissue residue technique, and by the i.v. injection of trypan blue. The former showed that the barrier had become permeable to D-sucrose, inulin, and L-glucose, substances that usually do not cross the barrier; the latter showed that the barrier had also become permeable to trypan blue, so that the brains of the comatose animals stained blue whereas normal brains did not. The brains of comatose animals uniformly showed cerebral edema on gross examination (their water contents were increased), and, on electron microscopic examination, their astrocytes were swollen, especially in pericapillary locations. The break in the barrier appears to be associated with the formation of cerebral edema, and implies both that substances that usually do not gain access to the brain parenchyma can now do so and that substances usually conserved, such as neurotransmitters, can now escape.

0895 CAN HEPATIC COMA BE CAUSED BY A REDUCTION OF BRAIN NORADRENALINE OR DOPAMINE? (Eng.) Zieve, L.; Olsen, R. L. (Veterans Admin. Hosp., Minneapolis, MN 55417). *Gut* 18(9):688-691; 1977.

The hypothesis that reduction in brain dopamine or norepinephrine causes hepatic coma was tested in male Sprague-Dawley rats. Intraventricular infusions of octopamine (3.2 mg over 290 min), which raised brain octopamine concentrations more than 20,000-fold, resulted in reduction in brain norepinephrine and dopamine by as much as 90% without affecting the alertness of activity of normal rats. As this reduction of brain catecholamines is much greater than any reported in hepatic coma, the values observed in experimental hepatic failure probably have no etiological significance for the encephalopathy that ensues.

0896 THE EFFECT OF GLUCAGON AND BLOOD TRANSFUSION ON HEPATIC CIRCULATION AND OXYGEN CONSUMPTION IN HEMORRHAGIC SHOCK. (Eng.) Lindberg, B.; Darle, N. (Sahlgrenska sjukhuset, S-413 45 Goteborg, Sweden). *J. Surg. Res.* 23(4):257-263; 1977.

Experiments were carried out to determine whether the injection of glucagon combined with blood transfusions could increase liver blood flow and oxygen consumption in the treatment of hemorrhagic shock. Ten pigs were subjected to a standard shock model, including two 30-min periods of hemorrhagic shock at 50 mm Hg. After the first period of shock, glucagon (50 μg/kg) was administered i.v., and the shed blood was transfused. Portal vein and hepatic artery flow as well as oxygen consumption were measured. Both periods of shock resulted in a marked decrease in blood flow as well as oxygen

consumption. Treatment with glucagon and blood transfusion resulted in an increase in hepatic artery flow to 300% of the preshock value, but no significant increase in the portal vein flow occurred compared with blood transfusion alone. The high hepatic artery flow caused a very high inflow of oxygen to the liver. In spite of this high inflow of oxygen, the liver oxygen consumption was not significantly higher than after blood transfusion alone. Glucagon administration to pigs in hemorrhagic shock does not increase portal vein flow, as it does in dogs.

0897 THE EFFECT OF DEXTRAN 40 AND BLOOD TRANSFUSION ON HEPATIC CIRCULATION AND OXYGEN CONSUMPTION IN HEMORRHAGIC SHOCK. (Eng.) Lindberg, B.; Darle, N. (Sahlgrenska sjukhuset, S-413 45 Goteborg, Sweden). *J. Surg. Res.* 23(4):264-273; 1977.

The effect of treatment with dextran 40 and blood transfusion on hepatic circulation and oxygen consumption was studied in pigs subjected to a standard model of hemorrhagic shock. The animals were bled to 50 mm Hg arterial pressure and kept at this pressure for 30 min, one group being treated with the shed blood and the other group with an equal volume of dextran 40. Thirty minutes after completion of the treatment, the animals were once again bled to 50 mm Hg and kept at this level for 30 min. During the first period of shock the flow in both vessels decreased to 53% of the baseline value, and the oxygen consumption fell to 41% of the initial values. After treatment with the shed blood, the flow in the hepatic artery and portal vein returned to preshock values, and the oxygen consumption was 39% higher than the initial values. After dextran 40 treatment, the flow in the hepatic artery was 91% higher than the initial value and the flow in the portal vein was 127% higher. In spite of these high flow values, the oxygen consumption was significantly lower in the dextran 40 group. In the second period of shock, flow values were reduced to approximately the same values as in the first period, but the oxygen consumption was significantly lower in the dextran 40 group than in the blood transfusion group. The hepatic circulation during shock is different in the pig compared with that in the dog and cat.

0898 THE ENHANCEMENT OF ADENYL CYCLASE BY STEROID THERAPY IN SHOCK. (Eng.) Jones, C. A.; McArdle, A. H.; Hinchey, E. J. (Montreal General Hosp., Montreal, Quebec H3G 1A4, Canada). *Surgery* 82(4):483-488; 1977.

Experiments were carried out in dogs to study the normal response of the second messenger system in splanchnic tissues to hormonal stimulation and to determine whether the hormonal response is altered by hemorrhagic shock. Hepatic and intestinal adenyl cyclase (AC) activity were measured after a single pulse injection of epinephrine (0.25 mg, i.v.) or glucagon (0.3 mg, i.v.) into normal dogs and into dogs subjected to hemorrhagic shock. Hemorrhagic shock abolished the increase in AC activity seen in normal animals following epinephrine and significantly reduced that induced by glucagon. These changes were reflected in the glucose production from the liver induced by these hormones. The response of AC to the *in vitro* addition of epinephrine or glucagon, as well as the nonspecific stimulator of AC, sodium fluoride, showed that it is the receptor site of the enzyme that is primarily affected by shock. The treatment of dogs with 30 mg/kg of methylprednisolone following the reinfusion of shed blood significantly improved the response of AC to epinephrine in both liver and intestine, and this improvement was reflected in the glucose production by the liver in response to the hormone. The hormonal sensitivity of the splanchnic second messenger system, which is reduced significantly in shock, appears to be markedly improved by the administration of pharmacological doses of steroids.

0899 CHANGES IN CARDIAC OUTPUT DISTRIBUTION AFTER LIVER DEARTERIALIZATION IN THE RAT. (Eng.) Lindell, B.; Aronsen, K. F. (Malmo General Hosp., Malmo, Sweden). *Acta Chir. Scand.* 143(4): 207-213; 1977.

The changes in cardiac output distribution were studied in male Wistar rats after hepatic artery ligation and additional ligament division. These procedures resulted initially in a marked decrease in the arterial blood supply to the liver, and this effect was still pronounced 4 weeks after the operation. The decrease in liver blood supply induced by the procedure was in part counterbalanced by arteriovenous shunting in the preportal region. A temporary redistribution of cardiac output with reduced fractions to colon and spleen was found 2 weeks after these operations; this finding may have been a result of diversion of arterial blood through arteriovenous shunts.

0900 MORPHOLOGICAL ALTERATIONS AND FUNCTIONAL CHANGES OF INTERHEPATOCELLULAR JUNCTIONS INDUCED BY BILE DUCT LIGATION. (Eng.) Metz, J.; Aoki, A.; Merlo, M.; Forssmann, W. G. (Anatomisches Institut der Universitat, Im Neuenheimer Feld 307, D-6900 Heidelberg, W. Germany). *Cell Tissue Res.* 182(3):299-310; 1977.

The effect of bile duct ligation on the intercellular junctions of hepatocytes was investigated in male Wistar rats. The features and the arrangement of the bile canaliculi and the zonulae occludentes were altered 48 hr after ligation, which caused an increase in intracanalicular pressure. The lumina of the bile canaliculi enlarged, and the microvilli disappeared. The array of the zonulae occludentes became irregularly shaped, the number of strands diminished, and interruptions of the strands occurred. One minute after the i.v. injection of horseradish peroxidase (10 mg/100 g), a leakage in the bile-blood barrier was detected. The gap junctions between the hepatocytes disappeared after bile duct ligation. The results show that

the zonulae occludentes are mobile structures that are changed by increased unilateral pressure. Due to their ultrastructural alterations, a leakage of the permeability barrier between physiological compartments occurs.

0901 CONTROL OF LIVER SIZE IN HEAT-ACCLIMATED HAMSTERS. (Eng.) Chayoth, R.; Krauthammer, N.; Winikoff, J.; Sod-Moriah, U. A. (Dept. Biology, Ben Gurion Univ. of the Negev, Beer Sheva, Israel). *J. Appl. Physiol.* 43(3):445-448; 1977.

To determine whether the decreased liver size seen in hamsters exposed to high ambient temperatures is controlled by cyclic AMP, or by other factors, the activity of DNA synthesis was correlated with cyclic AMP levels in the livers of hamsters maintained in a hot environment (35 ± 1 C) and of controls, maintained at 22 ± 1 C. In the heat-exposed animals, liver weight decreased more than did body weight. Therefore, liver weight constitutes a lower percentage of body weight during exposure to high ambient temperature. This change was not a result of dehydration, since water content of the whole body and of the liver was not altered during heat acclimation. However, changes in lactic dehydrogenase isozyme proportions indicated a higher rate of liver degradation during the first 2 weeks of heat exposure. These changes were accompanied by enhancement of DNA synthesis, which was elevated during the early period of heat exposure and later fell to the control levels. The enhanced DNA synthesis might be a result of a high rate of tissue regeneration, which probably takes place in the organ following the commencement of the degradative processes. Since the activity of DNA synthesis was negatively correlated with cyclic AMP levels, it is suggested that cyclic AMP plays some role in controlling hepatic DNA synthesis during heat acclimation.

0902 INFUSION TOMOGRAPHY OF THE GALLBLADDER: MECHANISM OF GALLBLADDER WALL OPACIFICATION IN EXPERIMENTAL ACUTE CHOLECYSTITIS. (Eng.) Moncada, R.; Cardoso, M.; Danley, R.; Rodriguez, J.; Kimura, K.; Pickleman, J.; Brandly, J. (Loyola Univ. Medical Center, Maywood, IL 60153). *Am. J. Roentgenol.* 129(4):587-590; 1977.

A canine model of acute cholecystitis was used to determine the mechanism of opacification of the gallbladder wall by infusion tomography. The dogs underwent cystic duct ligation or cystic duct and cystic artery ligation. About 48 hr later, ^{125}I-labeled meglumine and sodium diatrilzoate were infused i.v. to a dose of 2.2 ml/kg. In normal gallbladders, the majority of the radioactivity was demonstrated within the blood vessels. There was no radioactivity in the extravascular spaces. A large proportion of the active contrast material had been taken up within polymorphonuclear granulocytes. In inflamed gallbladders, there was also some radioactive contrast material in the vascular spaces. In the extravascular space, contrast material was present exclusively within polymorphonuclear granulocytes. Some of these WBC may have come out of the vessels due to vessel disruption, as hemorrhage was seen in a few acutely inflamed gallbladders. However, even when the vessels maintained their integrity, WBC containing contrast material were seen in the edematous walls, especially in the subserosa. The results indicate that in the normal gallbladder, labeled radiopaque contrast material remains within the blood vessels, where some is taken up by polymorphonuclear leukocytes. In the inflammed gallbladder, inflammatory cells containing labeled contrast material are found in the edematous tissues of the gallbladder wall. This, together with hyperemia and probably tissue-fluid equilibration of contrast material, seem to be responsible for the visualization of the gallbladder wall with infusion tomography.

0903 INCREASED BILIARY TREE PERMEABILITY PRODUCED IN RATS BY HEPATOACTIVE AGENTS. (Eng.) Peterson, R. E.; Fujimoto, J. M. (Univ. Wisconsin, Center for Health Sciences, Madison, WI 53706). *J. Pharmacol. Exp. Ther.* 202(3):732-739; 1977.

Changes in recovery of [^{3}H]sucrose or [^{3}H]mannitol administered by retrograde intrabiliary injection were compared between control groups and groups of Sprague-Dawley female rats treated with various drugs. After retrograde intrabiliary injection with 23.4 µl of the test substance, the bile duct cannula was occluded for 0.5 and 6 min (first experiment) or 0.5 and 3 min (other experiments) before bile flow was restarted, and individual drops were collected. In comparing the dose-response effects of dehydrocholate, cholate (C), and taurocholate (TC), it was shown that C and TC reduced [^{3}H]mannitol recovery beyond levels that could be attributed to the choleretic effect of these compounds. Similarly, in rats in which bile flow was reduced by hypothermia (31 C), C and TC reduced [^{3}H]mannitol recoveries, whereas dehydrocholate had no effect on the recovery. The authors conclude that C and TC increased biliary tree permeability as seen by the increase in [^{3}H]mannitol absorbed from the biliary tree. Female rats treated with ethynylestradiol (EE, 0.4 and 4 mg/kg/day) for 4 days had a reduced rate of bile flow. Comparable reductions in bile flow were achieved by the i.v. infusion of indocyanine green or by hypothermia. Since at these reduced bile flows only EE treatment reduced recovery of the markers, the authors conclude that EE increased biliary tree permeability. Also, EE-treated animals were less sensitive to the choleretic response but were more sensitive to the permeability-enhancing effect of various doses of TC than were control animals.

0904 HEPATIC EXCRETION OF CHOLECYSTOPAQUES INTRODUCED INTO THE CANINE JEJUNUM. (Eng.) Menuck, L.; Amberg, J. R.; Bates, M. (Dept. Radiology, Univ. California, San Diego, CA). *Invest. Radiol.* 12(1):106-108; 1977.

The biliary excretion of five oral cholecystopaques (calcium ipodate, sodium ipodate, sodium trypanoate,

iocetamic acid, and iopanoic acid) was measured following their introduction into the jejunum of bile fistula dogs. The mean maximal iodine concentration in the bile ranged from 2.2 ± 0.55 mg/ml following a dose of 33 mg of iodine/kg of iopanoic acid to 12.9 ± 1.4 mg/ml after the same dose of sodium ipodate. Cumulative excretion at the end of 3 hr was over five times greater with sodium ipodate in comparison to ipoanoic acid. The ability of the more water-soluble media to pass through the unstirred water layer of the intestine may explain these data.

0905 TECHNETIUM-99m-LABELED N-(2,6-DIMETHYLPHENYLCARBAMOYLMETHYL) IMINODIACETIC ACID (Tc-99m HIDA): A NEW RADIOPHARMACEUTICAL FOR HEPATOBILIARY IMAGING STUDIES. (Eng.) Ryan, J.; Cooper, M.; Loberg, M.; Harvey, E.; Sikorski, S. (Univ. Maryland Hosp., 22 S. Greene St., Baltimore, MD 21201). *J. Nucl. Med.* 18(10):997-1004; 1977.

An easily formulated, stable kit preparation of technetium-99m-labeled *N*-(2,6-dimethylphenylcarbamoylmethyl) iminodiacetic acid (^{99m}Tc HIDA), suitable for use in humans, was developed and tested in mice and dogs. The tracer was cleared rapidly from the blood and excreted predominantly by the liver in both species. In dogs, the hepatobiliary clearance of ^{99m}Tc HIDA was significantly greater than that of ^{14}C HIDA and ^{113}Sn HIDA. The LD_{50} for HIDA in mice, 168 mg/kg, exceeded the average human dose by a factor of 1,000 on a per-weight basis. Blood clearance curves for ^{99m}Tc HIDA in 12 normal subjects were biexponential with half-times of 4.6 ± 1.0 min and 31.5 ± 7.0 min, and cumulative 90-min urine samples contained 14.2 ± 1.8% of the injected dose. Images in normal subjects and nonjaundiced patients showed rapid concentration of tracer by the liver, and activity was present within the biliary system in 10-20 min. In jaundiced patients, the tracer blood clearance was delayed and urinary excretion increased, but intestinal activity, indicating biliary patency, was imaged in those patients without complete focal obstruction of the common duct. ^{99m}Tc HIDA is a nontoxic radiopharmaceutical useful for clinical evaluation of hepatobiliary disorders in humans.

0906 INTERACTION OF BROMOSULFOPHTHALEIN WITH MITOCHONDRIAL MEMBRANES--INHIBITION OF RESPIRATION. (Eng.) Burr, R.; Schwenk, M.; Pfaff, E. (Inst. Toxicology, Univ. Tubingen, D-7400 Tubingen, W. Germany). *Biochem. Pharmacol.* 26(6):461-466; 1977.

The kinetics of mitochondrial respiratory inhibition produced by BSP were studied. BSP reversibly inhibited mitochondrial respiration. The most sensitive was state 3 respiration (Ki about 3 nmol/mg protein independent of substrate). At higher concentrations (20-100 nmol/mg protein), state 4 respiration and uncoupled respiration were also inhibited. This inhibition was substrate dependent. With succinate, inhibition appeared to be noncompetitive at low concentrations and competitive above 1 mM succinate (half-maximal inhibition at 9-17 nmol/mg protein, dependent on succinate concentration). Substrate permeation did not seem to be the only sensitive step in oxidation, as was deduced from similar results obtained on glycerol phosphate respiration. Using artificial electron shunts and difference spectroscopy, individual dehydrogenases were found to be probable sites of BSP action. It is suggested that BSP acts via the electrostatic effects of the increased negative surface charge, making dehydrogenases less accessible for their substrates.

0907 INHIBITION BY BROMSULPHTHALEIN OF THE BILIARY EXCRETION OF ITS GLUTATHIONE CONJUGATE. (Eng.) Gregus, Z.; Fischer, E.; Varga, F. (Univ. Medical Sch., Szigeti u. 12, H-7643 Pecs, Hungary). *Biochem. Pharmacol.* 26(20):1951-1952; 1977.

The importance of the competition between BSP and its glutathione conjugate (BSP-GSH) during the biliary excretion of the two substances was investigated in rats. The concentration of BSP in the liver was much higher than that of BSP-GSH. In bile duct-ligated rats, BSP-GSH proved to be a very weak inhibitor of the hepatic uptake of BSP. Similarly, BSP depressed the hepatic concentration of BSP-GSH significantly ($p<0.01$) only at a dose of 120 µmol/kg (as compared with a dose of 60 µmol/kg, i.v.). However, 60 µmol/kg of BSP did not affect the hepatic uptake of BSP-GSH, although the same dose of BSP markedly depressed the biliary excretion of BSP-GSH. These results suggest that competition between BSP and BSP-GSH for hepatic uptake may not be significant in the overall transport of BSP or BSP-GSH from blood to bile. BSP-GSH (60-120 µmol/kg) did not significantly depress the biliary excretion of BSP, but the excretion rate of BSP-GSH was markedly depressed by a dose as low as 15 µmol/kg of BSP. A significant inhibition of BSP-GSH excretion was observed in the first 15 min of bile collection with a simultaneous administration of 7.5 µmol/kg of BSP. The excretion of BSP-GSH in the following 30 min of bile collection masked this inhibition. The results indicate that the inhibitory effect of free BSP on BSP-GSH excretion may have an important role in the transport rate of total dye excretion.

0908 PHARMACOKINETIC ASPECTS OF SULFOBROMOPHTHALEIN TRANSPORT IN CHRONICALLY CARBON TETRACHLORIDE-INTOXICATED RATS. (Eng.) Iga, T.; Sugiyama, Y.; Yokota, M.; Tomono, Y.; Awazu, S.; Hanano, M. (Faculty Pharmaceutical Sciences, Univ. Tokyo, Bunkyo-ku, Tokyo 113, Japan). *Biochem. Pharmacol.* 26(20):1867-1875; 1977.

To elucidate the factors affecting the hepatic transport of BSP in pathological conditions, the kinetics (i.e., binding activities of plasma and hepatic cytoplasmic proteins, and glutathione S-transferase activity of the Y-fraction) of the BSP disappearance from the blood and appearance in the bile were studied in rats chronically intoxicated with carbon tetrachloride (0.1 ml/100 g, s.c., twice weekly, 8-9 weeks). Although plasma protein albumin concentration decreased during chronic in-

toxication, no quantitative difference was shown in the high affinity binding site (K_1). A significant decrease was shown in the cytoplasmic protein concentration for the Y- and Z- fractions in intoxicated rats; no significant difference was observed in the number of binding sites for both fractions, but their binding constants decreased. Glutathione S-transferase activity and glutathione (GSH) content were not altered, although a decrease in conjugating activity per rat was expected. The time course for the plasma disappearance and biliary excretion of BSP showed a remarkable delay after CCl_4 intoxication, while no difference was shown in the bile flow rate. It is concluded that hepatic blood flow might play a primary role in the initial plasma disappearance of BSP and that the decrease in the Y-fraction binding activity might explain the decrease in BSP uptake rate into the liver that was observed in intoxicated rats.

0909 TRANSFER AND REOXIDATION OF REDUCING EQUIVALENTS AS THE RATE-LIMITING STEPS IN THE OXIDATION OF ETHANOL BY LIVER CELLS ISOLATED FROM FED AND FASTED RATS. (Eng.) Cederbaum, A. I.; Dicker, E.; Rubin, E. (Mount Sinai Sch. Medicine, City Univ. New York, New York, NY 10029). *Arch. Biochem. Biophys.* 183(2):638-646; 1977.

Factors regulating the oxidation of ethanol were studied in liver cells isolated from fed and fasted Sprague-Dawley rats. The rate of ethanol oxidation was greater in liver cells from fed rats than from fasted rats. Inhibitors of the malate-aspartate shuttle decreased the rate of ethanol oxidation, suggesting that this shuttle contributes to the reoxidation of cytosolic NADH produced during the oxidation of ethanol. The greater inhibition of ethanol oxidation by antimycin than by rotenone suggested that the α-glycerophosphate shuttle also plays an important role in transporting reducing equivalents. The components of the malate-aspartate and α-glycerophosphate shuttles stimulated ethanol oxidation to a greater extent in liver cells from fasted rats than those from fed rats, suggesting that in the fasted state ethanol oxidation is regulated by the intracellular concentrations of substrate shuttle components, which transfer reducing equivalents into the mitochondria. Therefore, uncoupling agents, which stimulate oxygen consumption, do not stimulate ethanol oxidation, and concentrations of antimycin that depress oxygen uptake are much less effective in decreasing ethanol oxidation. By contrast, in liver cells from fed rats, the rate of ethanol oxidation was increased by uncoupling agents. Such stimulation was not observed when cells were prepared in the absence of albumin, probably due to leakage of shuttle substrates that leads to abnormally low intracellular levels. When the shuttle substrates were added back to these preparations, uncouplers were effective in stimulating the rate of ethanol oxidation beyond the stimulation produced by the shuttle substrates alone. Thus, under conditions of sufficient intracellular levels of the intermediates of the substrate shuttles, ethanol oxidation is regulated by the capacity of the mitochondrial respiratory chain to reoxidize reducing equivalents generated by the alcohol dehydrogenase reaction.

0910 THE EFFECTS OF ACUTE ETHANOL FEEDING AND OF CHRONIC BENFLUOREX ADMINISTRATION ON THE ACTIVITIES OF SOME ENZYMES OF GLYCEROLIPID SYNTHESIS IN RAT LIVER AND ADIPOSE TISSUE. (Eng.) Pritchard, P. H.; Bowley, M.; Burditt, S. L.; Cooling, J.; Glenny, H. P.; Lawson, N.; *et al.* (Univ. Hosp. and Medical Sch., Clifton Blvd., Nottingham NG7 2UH, England). *Biochem. J.* 166(3):639-642; 1977.

The effects of ethanol and of 1-(3-trifluoromethylphenyl)-2-[*N*-(2-benzoyloxyethyl)amino]propane or S-780 (benfluorex) on the activities of some of the enzymes of glycerolipid synthesis were investigated in the livers and adipose tissue of male Wistar rats. The rats were treated for 5 days with benfluorex (50 mg/kg) or with suspending medium (controls). They were then intubated with an acute intoxicating dose of ethanol (5 g/kg) or with glucose of equivalent energy content. Ethanol treatment of the control rats that had not received benfluorex specifically increased the hepatic activity of the soluble phosphatidate phosphohydrolase by about fivefold in 6 hr. The equivalent increase in the benfluorex-treated rats was about twofold. The activities of the other enzymes tested (liver glycerol phosphate acyltransferase; microsomal phosphatidate phosphohydrolase, phosphatidate cytidylyltransferase, choline phosphotransferase, and diacylglycerol acyltransferase; and adipose glycerol phosphate acyltransferase, total phosphatidate phosphohydrolase, ATP→Coenzyme A-dependent synthesis of acylalkylglycerol, and ATP→Coenzyme A-dependent synthesis of acylalkylglycerol from hexadecylglycerol) were not significantly altered. The results are compatible with the hypothesis that changes in the activity of the soluble phosphatidate phosphohydrolase contribute to the control of the metabolism of fatty acids and to their incorporation into hepatic triacylglycerols.

0911 EFFECTS OF CLOFIBRATE ON ETHANOL-INDUCED MODIFICATIONS IN LIVER AND ADIPOSE TISSUE METABOLISM: ROLE OF HEPATIC REDOX STATE AND HORMONAL MECHANISMS. (Eng.) Savolainen, M. J.; Jauhonen, V. P.; Hassinen, I. E. (Dept. Medical Biochemistry, Univ. Oulu, Kajaanintie 52 A, SF-90220 Oulu 22, Finland). *Biochem. Pharmacol.* 26(5):425-431; 1977.

Mechanisms by which clofibrate decreases the development of acute alcoholic fatty liver were studied in rats, especially in regard to hepatic redox state and hormonal regulation of carbohydrate and lipid metabolism. A partial inhibition of the ethanol-induced increase in cytosolic NADH/NAD ratio was observed. As a result, in clofibrate-treated rats (600 mg/kg/day, 9 days, s.c.) hepatic sn-glycerol-3-phosphate (α-GP) concentration during ethanol oxidation also remained at the same level as in normal control rats. Clofibrate treatment prevented the ethanol-induced increase in the adi-

pose tissue cyclic AMP and plasma free fatty acid concentrations. Hepatic concentrations of coenzyme A (Co-A)-SH, acetyl-CoA, and long-chain acyl-CoA were markedly increased by clofibrate treatment. Plasma insulin concentration was decreased in clofibrate-treated rats, which also showed an impaired glucose tolerance. The results show that clofibrate is able to restrict the availability of substrates (α-GP and fatty acids) for hepatic triglyceride synthesis *in vivo*. In addition, it is concluded that the partial inhibition of ethanol-induced fatty liver by clofibrate may result from the enhancement of the oxidation pathway of fatty acid metabolism, as suggested by the enormous increase in hepatic content of CoA-SH and its derivatives.

0912 THE MECHANISM OF ENHANCEMENT OF RAT LIVER TRYPTOPHAN PYRROLASE ACTIVITY BY ACUTE ETHANOL ADMINISTRATION. (Eng.) Badawy, A. A. B.; Evans, M. (Univ. Hosp. Wales, Cardiff CF4 7XB, Wales). *Biochem. Soc. Trans.* 5(4):1035-1036; 1977.

The mechanism by which rat liver tryptophan pyrrolase activity is enhanced by acute ethanol administration was investigated by determining the half-life of the enzyme after acute ethanol administration (5 ml/kg, i.p. as a 25% volume/volume solution) in intact male Wistar rats. Ethanol significantly ($p<0.001$) enhanced the holoenzyme and total pyrrolase activities by 2.35- and 1.78-fold, resp., and increased the ratio of holoenzyme/apoenzyme activity from a control value of 0.74 to 1.45. The half-life of the total enzyme in ethanol-treated rats was 3.94 hr, which represented a 73% increase in the stability of the pyrrolase by ethanol. This finding provides further support for the involvement of tryptophan in the ethanol effect.

0913 THE MECHANISM OF INHIBITION OF RAT LIVER TRYPTOPHAN PYRROLASE ACTIVITY BY CHRONIC ETHANOL ADMINISTRATION. (Eng.) Evans, M.; Badawy, A. A. B. (Univ. Hosp. Wales, Cardiff CF4 7XB, Wales). *Biochem. Soc. Trans.* 5(4):1037-1038; 1977.

The mechanism by which the chronic administration of ethanol inhibits rat liver tryptophan pyrrolase activity was investigated by determining the effect of chronic ethanol, sucrose, or water treatment on cortisol-induced pyrrolase activity. Male Wistar rats received one of the following three drinking fluids for 28-31 days: water, sucrose (9%, weight/volume), or ethanol (5% volume/volume for 2 days, 7.5% for 2 further days, and 10% thereafter). Some rats also received cortisol acetate (20 mg/kg, i.p.) 4 hr before sacrifice. Chronic ethanol or sucrose administration inhibited the total pyrrolase activity by 35% but had no effect on that of the holoenzyme; the apoenzyme was therefore the form specifically inhibited by either treatment (by 80%, $p<0.001$). Cortisol was equally effective in inducing the enzyme activity in both chronic ethanol-treated and control rats, suggesting that ethanol does not inhibit the synthesis of the apoenzyme. The cortisol effect in rats treated chronically with sucrose was, however, lower by 24-34% than that in controls. The results suggest that sucrose is not a suitable control treatment in studies of the effects of ethanol on liver or brain tryptophan metabolism.

0914 IN VIVO STUDIES OF AMINO ACID METABOLISM IN RAT INTESTINE AND LIVER. (Fre.) Demigne, C.; Aufrere, J.; Remesy, C. (Station de Physiopathologie de la Nutrition, I.N.R.A., Theix, Saint Genes Champanelle, 63110 Beaumont, France). *Ann. Biol. Anim. Biochim. Biophys.* 17(4):615-620; 1977.

Liver uptake and intestinal metabolism of amino acids in rats during absorption of a diet high in casein (13%) was studied. The diet fed to Sherman rats, conditioned to absorb their food in 8 hr, consisted of 79% wheat starch, 2% corn oil, 5% minerals, and 1% vitamins in addition to the casein. Animals were anesthetized with Nembutal (40 mg/kg, s.c.) 4 hr after they began eating, and blood was removed for amino acid analysis from the portal vein and artery or from the hepatic and portal veins. Livers were then removed, minced, and centrifuged. Alanine in the portal vein came from casein hydrolysis, from transamination of glutamic and aspartic acids in the intestinal cells, and from deaminated glutamine. Amino acid release into the portal vein was compared with overall liver balance, taking into account the flow rate of the hepatic artery. The uptake of alanine, glycine, and aromatic amino acids by the liver was high (78%, 82%, and 64-70% of the total alanine, glycine, and aromatic acids in the administered casein, resp.), while that of serine and threonine was low (49% and 43%, resp.). Only a small fraction of branched-chain amino acids was taken up by the liver (about 20%); the main uptake of these occurred in the periperhal tissues.

0915 THE ISOLATION AND FURTHER CHARACTERIZATION OF THE BILIRUBIN TETRAPYRROLES IN BILE-CONTAINING HUMAN DUODENAL JUICE AND DOG GALL-BLADDER BILE. (Eng.) Gordon, E. R.; Chan, T. H.; Samodai, K.; Goresky, C. A. (Montreal General Hosp., 1650 Cedar Ave., Montreal, Quebec H3G 1A4, Canada). *Biochem. J.* 167(1):1-8; 1977.

A simple reproducible method for the isolation of the native bilirubin tetrapyrroles from bile-containing human duodenal juice and dog gallbladder bile is described, and the tetrapyrroles are further characterized. Bilirubin and its conjugates are extracted from bile or bile-containing duodenal juice into chloroform containing 10 mM tetraheptylammonium chloride. The intact bilirubin tetrapyrroles are then separated by thin-layer chromatography. Structural elucidation was made after coupling of the individual pigments with diazonium salts. Four azopigments were detected: azopigment α_0 or dipyrrolic azobilirubin, azopigment δ or dipyrrolic azobilirubin monoglucuronide, azopigment α_3 or dipyrrolic azobilirubin monoglucoside, and, from dog gallbladder bile, azopigment α_2. The last conjugate required further verification of its structure. After methanolysis, it was shown by

combined gas-liquid chromatography-mass spectrometry to contain xylose in a 1:1 molar ratio with the azopigments of bilirubin. Human bile contained 86% bilirubin monoglucuronide, 7% bilirubin monoglucuronide monoglucoside diester, 4% bilirubin monoglucuronide, and 3% bilirubin. Dog gallbladder bile had a considerably different composition; it contained 47% bilirubin diglucuronide, 40% bilirubin monoglucuronide monoglucoside diester, 8% bilirubin monoglucuronide, 4% bilirubin diglucoside, 1-2% bilirubin, and traces of conjugates containing xylose. The total bilirubin content and proportions of the conjugates did not change in bile that was frozen and stored at -20 C under N_2, whereas in the chloroform/tetraheptylammonium chloride extract, similarly stored, total pigment was slowly lost and the diglucuronide conjugate converted into the monoglucuronide.

0916 SYNTHESIS AND ENTEROHEPATIC CIRCULATION OF BILE SALTS. (Eng.) Hanson, R. F.; Pries, J. M. (Univ. Minnesota Hosp., Minneapolis, MN). *Gastroenterology* 73(3):611-618; 1977.

A review is presented of the present knowledge of bile salt synthesis, secretion, absorption by the intestine, and removal by the liver. The pathways for bile salt synthesis, bile acid conjugation, and sulfation, and the role of carrier proteins in bile acid synthesis are discussed. Interaction of bile salts with hepatic enzymes such as 3-hydroxy-3-methylglutaryl-CoA reductase, 7-hydroxylase, and 12-hydroxylase is reviewed. It is suggested that the uptake of bile salts is mediated by a Na^+-dependent carrier transport mechanism.

0917 SEX-LINKED DIFFERENCES IN BILE ACID METABOLISM OF GERMFREE RATS. (Eng.) Eyssen, H.; Smets, L.; Parmentier, G.; Janssen, G. (Rega Inst. Medical Res., Univ. Leuven, Minderbroedersstraat 10, B-3000 Leuven, Belgium). *Life Sci.* 21(5):707-712; 1977.

To investigate the influence of sex on bile acid composition, sulfated and unsulfated bile acids were quantitated in male and female germfree rats. Gas chromatography and mass spectrometry revealed that β-muricholic acid and cholic acid were the major bile acids in both sexes; in addition, 3β-hydroxy-5-cholenoic acid, chenodeoxycholic acid, α-muricholic acid, allochenodeoxycholic acid, and allocholic acid were present. β-Muricholic and cholic acids accounted for 61.4% and 22.7%, resp., of total bile acids in the small intestine of males; females had 38.9% of β-muricholic acid and 50% of cholic acid. In females, the bile acid sulfate fraction increased from 1.1% in the small intestine to 22.3% in the large intestine; in males these values were 0.2% and 1.7%, resp. A considerable increase in the relative amounts of allochenodeoxycholic and allocholic acids was observed in the cecum and large intestine of the female rat, where more than 70% of these substances was in the bile acid sulfate fraction. In males, these allobile acids were mainly in the unsulfated fraction, and their relative amounts did not increase in the large intestine. This study shows important sex-linked differences in the relative amounts and sulfation of bile acids in germfree rats.

0918 BILE ACID BINDING BY 100 000 g SUPERNATANTS FROM RAT LIVER. (Eng.) Strange, R. C.; Nimmo, I. A.; Percy-Robb, I. W. *In: Bile Acid Metabolism in Health and Disease. Proceedings of the IV Bile Acid Meeting held at the Hilton Hotel, Basel, Switzerland, October 11-12, 1976.* University Park Press (Baltimore, MD): 125-132; 1977.

The binding of cholic acid, glycocholic acid, chenodeoxycholic acid, and lithocholic acid to rat liver 100,000 x *g* supernatants was demonstrated using equilibrium dialysis. Rectangular hyperbolae fitted the binding data for chenodeoxycholic acid, lithocholic acid, and glycocholic acid, suggesting that binding could be explained in terms of single saturable components. The binding data for cholic acid were better fitted by a straight line plus a rectangular hyperbola, suggesting the presence of at least two different types of binding sites. The binding characteristics of the bile acids suggest that the binding components are involved in bile acid transport. When mixtures of [^{14}C]lithocholic acid and liver supernatants were eluted from columns of Sephadex G-75, a prominent peak of [^{14}C]lithocholic acid appeared with proteins of molecular weight approximately 40,000. A second, smaller peak of [^{14}C]lithocholic acid was eluted with proteins of molecular weight approximately 10,000.

0919 HYDROXYLATIONS IN BIOSYNTHESIS AND METABOLISM OF BILE ACIDS CATALYZED BY RECONSTITUTED SYSTEMS FROM RAT LIVER MICROSOMES. (Eng.) Cottman, J.; Danielsson, H.; Hansson, R.; Wikvall, K. *In: Bile Acid Metabolism in Health and Disease. Proceedings of the IV Bile Acid Meeting held at the Hilton Hotel, Basel, Switzerland, October 11-12, 1976.* University Park Press (Baltimore, MD): 1-10; 1977.

Several hydroxylations in the biosynthesis and metabolism of bile acids were studied in reconstituted systems from rat liver microsomes consisting of cytochrome P-450 and NADPH-cytochrome P-450 reductase at different stages of purification. A partial separation of 5β-cholestane-3α,7α-diol 12α- and 26-hydroxylase activities was obtained by polyethylene glycol fractionation of cholate-solubilized cytochrome P-450 followed by diethylaminoethyl (DEAE)-cellulose chromatography. These two hydroxylase activities differed not only from each other but also from other hydroxylase activities such as taurodeoxycholate 7α-hydroxylase activity and laurate 11- and 12-hydroxylase activities. Cholesterol and cholestanol 7α-hydroxylase activity was compared with 5β-cholestane-3α,7α-diol 12α- and 26 hydroxylase activities. By polyethylene glycol and ammonium sulfate fractionation, a partial separation of these three activities was obtained. The possibility that some specificity

also resided in the NADPH-cytochrome P-450 reductase was tested with reductase preparations of different purity. In contrast to several other hydroxylations including 12α-hydroxylation of 5β-cholestane-3α,7α-diol, 26-hydroxylation of 5β-cholestane-3α,7α,12α-triol and 7α-hydroxylation of taurodeoxycholic acid, which were all stimulated by the different preparations, 25-hydroxylation of 5β-cholestane-3α,7α,12α-triol and 7α-hydroxylation of cholesterol were stimulated to a greater extent by the less pure preparations than by the purer preparations. The results indicate the participation of several cytochromes P-450 in the hydroxylations occurring in bile acid biosynthesis and metabolism and point to the possibility that other factors present in less pure preparations of NADPH-cytochrome P-450 reductase also may play a role in imparting specificity to the hydroxylations.

0920 EXPERIMENTAL CHOLESTASIS BY DIBUCAINE AND HARMALINE: EFFECTS ON BILE FLOW AND HEPATIC TRANSPORT OF BILE ACIDS, ETHACRYNIC ACID AND OUABAIN. (Ger.) Peterlik, M. (Institut fur allgemeine und experimentelle Pathologie, Universitat Wien, Wahringer Strasse 13, A-1090 Vienna, Austria). *Wien. Klin. Wochenschr.* 89(14):494-501; 1977.

Dibucaine- and harmaline-induced experimental cholestasis and the effects of these substances on bile secretion and active transport of bile acids, ethacrynic acid and ouabain were studied in the isolated liver of adult Sprague-Dawley rats. Both dibucaine (0.5-1.6 mM) and harmaline (1.7-4 mM) inhibited bile secretion; a nearly linear dose-dependence effect was found for both substances. The effect of dibucaine was fully reversible, but bile secretion failed to reach the normal level after inhibition with harmaline at the highest concentration (4 mM), which may indicate partially irreversible damage of the secretory function of the liver. Both substances caused only a slight reduction in the oxygen consumption of the isolated liver. While typical signs of intrahepatic cholestasis (dilatation of the canaliculi) were seen, the ultrastructure of the liver was completely intact, and no signs of nonspecific liver damage (swelling of mitochondria or of the endoplasmic reticulum) were seen, which indicates that the cholestasis was not due to a nonspecific hepatotoxic effect. The transport of taurocholic acid into the isolated liver was influenced only slightly by dibucaine (1.6 mM) and harmaline (4 mM), but dibucaine inhibited the influx of cholic acid, ethacrynic acid, and ouabain by 60%, 58%, and 90%, resp., and harmaline caused inhibitions by 60%, 82%, and 86%, resp. The findings indicate that harmaline and dibucaine inhibit taurocholate transfer only to the extent of the Na^+-dependent fraction, and that drug-membrane interactions, primarily on the sinusoidal surface, but possibly also at the canalicular pole of the hepatocytes, are responsible for the impairment of basal and stimulated bile secretion. The perfusion model is suitable for the experimental study of intrahepatic cholestasis, of the influence of the hepatocyte membrane functions on canalicular bile secretion, and of the possible pathogenetic role of these functions in intrahepatic cholestasis.

0921 GLUCOCORTICOID INFLUENCE ON MORPHOLOGICAL DIFFERENTIATION AND BILE ACID METABOLISM IN RAT LIVER CELL CULTURE. (Eng.) Lambiotte, M. *In: Bile Acid Metabolism in Health and Disease. Proceedings of the IV Bile Acid Meeting held at the Hilton Hotel, Basel, Switzerland, October 11-12, 1976.* University Park Press (Baltimore, MD): 33-47; 1977.

The influence of dexamethasone (dex) on morphological differentiation and bile acid metabolism was studied in primary cell cultures of rat fetal liver, in monolayers of adult rat hepatocytes, and in a cloned strain of a cultivated "minimal deviation" rat hepatoma. Labeled cholic or deoxycholic acid (51 and 52 mCi/mmol, resp.) were added to the cultures, and the various metabolites appearing in the medium were tentatively determined using thin-layer chromatography (TLC), scanning, and autoradiography. In fetal liver cultures, dex (1.9×10^{-6} M) induced the appearance of bile canaliculi after 3 or 4 days, towards which the hepatocytes seemed to retain some polarity. Cholate and deoxycholate were conjugated mainly to taurine, with traces of glycine conjugates in the treated group; the conjugation was retained longer in the presence of dex. Deoxycholate was partially transformed into taurocholate, free cholate, and traces of glycocholate, but the hydroxylation was hardly noticeable in controls. The adult hepatocyte monolayers remained healthy longer in the presence of dex, whereas cells in the control cultures degenerated in 4-6 days. In the two experimental groups, deoxycholate was transformed into taurocholate, taurodeoxycholate, glycocholate, glycodeoxycholate, and free cholate. The proportion of glycine conjugates was higher than in the fetal liver culture, especially in the control group, and the hydroxylation products were more abundant in the presence of dex. In the hepatoma monolayers, cholate conjugation was obtained after only 2 days in dex, and deoxycholate conjugation was markedly increased by the hormone. Deoxycholate hydroxylation was noticed only in the treated plates; the main induced product had a relative front in TLC migration intermediate between glycocholate and glycodeoxycholate. The results provide good evidence for a glucocorticoid induction, not only of bile excretory pathways, but also of the 7α-hydroxylation system of deoxycholic acid, which could be important *in vivo* during the functional maturation of rat fetal liver.

0922 THE HEPATOTOXICITY OF LITHOCHOLIC ACID IN MALE MICE. (Eng.) Taylor, W.; Lesna, M. (Medical Sch., Univ. Newcastle upon Tyne, Newcastle upon Tyne NE1 7RU, England). *Br. J. Pharmacol.* 61(1):133P-134P; 1977.

The mechanism by which lithocholic acid (LA) causes intrahepatic cholestasis and early damage to the bile canaliculi in rodents was studied in Balb/c mice by determining the cholesterol and bile acids

in the gallbladder bile and by observing the development of hepatic lesions after the administration of LA (8 mg/day by gavage). Cholesterol was determined colorimetrically, and bile acids by gas chromatography-mass spectrophotometry, in animals killed 0.5, 1.5, 2.5, and 3.5 days after the start of treatment. The biliary cholesterol concentrations rapidly increased while total bile acids first increased and then decreased, causing a dramatic decrease in the total bile acid/cholesterol ratio. Between 1.5 and 2.5 days there was a large decrease in cholic acid concentration in the bile; both LA and 3α,6β-dihydroxy-5β-cholanoic acid, which were not detectable in control bile, appeared in increasing amounts. These changes preceded the appearance of liver damage, which was well established after 2.5 days. The livers of LA-treated animals showed necrotic areas of irregular distribution with some dilatation of centrilobular venules and inflammatory and neutrophil infiltration. LA may exert its hepatotoxic effect in mice by altering hepatic cholesterol and bile acid biosynthesis.

0923 BILE ACID CONTROL OF BILIARY CHOLESTEROL SECRETION IN MAN. (Eng.) Schersten, T.; Lindblad, L. *In: Bile Acid Metabolism in Health and Disease. Proceedings of the IV Bile Acid Meeting held at the Hilton Hotel, Basel, Switzerland, October 11-12, 1976.* University Park Press (Baltimore, MD): 175-181; 1977.

The interrelationships between biliary cholesterol secretion and the secretion of bile acids and lecithin were studied during depletion of the bile acid pool and during duodenal administration of cholic or chenodeoxycholic acid (1.0 g/hr, 4 hr) in 13 patients 7-12 days after cholecystectomy for uncomplicated gallstone disease. The relation between bile acid and cholesterol secretion rates was curvilinear during bile acid pool depletion ($r=0.70$) and during cholic acid secretion ($r=0.86$). The lecithin secretion rate was significantly correlated to the secretion rate of cholesterol under all the experimental conditions. These correlations were stronger at low bile acid secretion rates giving bile supersaturation in cholesterol. The results suggest that the biliary secretion rate of lecithin plays a determining role for the mass of secreted cholesterol and that the importance of lecithin is different at different rates of bile acid secretion.

0924 NUTRITIONAL AND HORMONAL CONTROL OF LUNG AND LIVER FATTY ACID SYNTHESIS. (Eng.) Kumar, S. (New Jersey Medical Sch., Newark, NJ 07103). *Arch. Biochem. Biophys.* 183(2):625-637; 1977.

The effect of a high-fructose diet on the levels of fatty acid synthesizing enzymes was investigated in male Sprague-Dawley rats, and extensive studies on the nutritional and hormonal control of fatty acid synthesis in the mammalian lung were carried out. During starvation and in streptozotocin-induced diabetes, the total activities of rat lung acetyl coenzyme A (CoA) carboxylase and fatty acid synthetase were reduced to one-third of the normal values. Refeeding the starved animals or the administration of insulin to diabetic animals restored the levels to the original values. The insulin effect was dose- and time-dependent. These data contrast with those in the liver, where a 30- to 50-fold depression of these enzymes was observed in the diabetic state, and the administration of insulin was actually followed by doubling of the activity over normal controls. A fat-free high-fructose diet (containing 60% fructose by weight) enhanced the activities of liver enzymes three- to sixfold over the values of controls on laboratory diet but had no effect on the lung enzymes. Long-term feeding of the fructose diet also increased the activities of liver enzymes from diabetic animals to twice the value of normal controls on laboratory diet. Insulin administration to fructose-fed diabetic animals restored the enzyme activities to those obtained with fructose-fed normal controls. However, the stimulation of lung enzymes of diabetic animals was effected either by fructose or by insulin. Antigen-antibody titrations and measurements of the rate of protein synthesis showed that the increased activity of the lung and liver fatty acid synthetase is due to enhanced content rather than increased specific activity. These data suggest that the effects of insulin or fructose on fatty acid synthesizing enzymes are mediated through intermediate(s) whose concentration is affected in the experimental diabetes. Furthermore, all tissues may not have stringent insulin requirements, since the lung enzymes can be stimulated by fructose alone.

0925 INCREASED LIVER CALCIUM AFTER CALCIUM OR MILK GAVAGE IN RATS. (Eng.) Meyer, R. A., Jr.; Meyer, M. H. (Sch. Dentistry, Marquette Univ., 604 North 16th St., Milwaukee, WI 53233). *Experientia* 33(4):546-547; 1977.

Experiments were carried out in male SPD rats to determine whether there is a change in liver calcium content during times of known calcitonin secretion. The liver calcium level was raised significantly at 0.5 hr ($p<0.01$) and at 1 hr ($p<0.05$) after gavage with milk (2 ml/100 g) and at 1 and 2 hr ($p<0.05$ and 0.01, resp.) after gavage with 300 mM $CaCl_2$ (2 ml/100 g), in comparison with that in controls that received either sham gavage or a deionized water gavage. Plasma calcium and phosphate were not significantly altered by the milk gavage, but were significantly affected by $CaCl_2$ gavage. The lack of change in plasma calcium, liver weight, and extracellular fluid content indicated that the increased liver calcium seen after the milk meal must represent increased intracellular calcium. Since the increase in liver calcium for the whole liver (0.27 µmol/100 g body weight) was quite small relative to the calcium content of the milk gavage (60 µmol/100 g body weight), it seems that the liver is not an important storage site for plasma calcium and that physiological stimuli of calcitonin secretion do not result in as large an increase in liver calcium as reported following the injection of the hormone.

0926 LIVER ENZYME INDUCTION BY THE ANION EXCHANGER RESIN, DOWEX 1 x 2, IN THE RAT. (Eng.) Beyhl, F. E.; Granzer, E. (Hoechst AG., Postfach 800320, D-6230 Frankfurt am Main 80, W. Germany). *Experientia* 33(9):1194-1195; 1977.

The nature of bile acid sequestration by the anion exchange resin, Dowex 1 x 2, was investigated by determining whether it induces the biosynthesis of microsomal drug metabolizing liver enzymes in male Wistar rats. The rats received the resin (500 mg/kg/day) by stomach tube for 8 days. This treatment led to liver enlargement and significant increases in liver acid phosphatase, glucose-6-phosphate dehydrogenase, malic enzyme, catalase, neotetrazolium reductase, NADPH:cytochrome c oxidoreductase, and aminopyrin demethylase ($p<0.001$, $p<0.001$, $p<0.0005$, $p<0.0005$, $p<0.0005$, $p<0.005$, and $p<0.0005$, resp.). The mitochondrial enzymes, glutamate and succinate dehydrogenases; mitochondrial cytochrome c reductase; and most of the soluble enzymes, α-glycerophosphate, malate, isocitrate, and 6-phosphogluconate dehydrogenases and the transaminase, were unchanged. Therefore, Dowex 1 x 2 treatment does not influence mitochondrial and cytosolic metabolisms, or at most, only parts of them. The sequestration of bile acids by anion exchange resin treatment with Dowex 1 x 2 mimics the biochemical effects of clofibraté- and phenobarbital-like drugs in the liver.

0927 THE EFFECT OF LEVAMISOLE ON PHOSPHODIESTERASE ACTIVITY. (Eng.) Constantopoulos, A.; Kafasi, V.; Doulas, N.; Liakakos, D.; Matsaniotis, N. (Aghia Sophia Children's Hosp., Athens, Greece). *Experientia* 33(3):395-396; 1977.

To determine whether levamisole acts by modifying nucleotide levels through activation of phosphodiesterase, the activity of this enzyme was estimated in mouse liver homogenates in the presence and absence of levamisole (10^{-2}-10^{-7} M). Phosphodiesterase activity was 1,394 and 1,399 nmol/mg protein/30 min in the presence and absence of the anthelmintic drug, resp. These data demonstrate that levamisole does not modify nucleotide levels via the activation of phosphodiesterase.

0928 DRY WEIGHT, TRIGLYCERIDES CONTENT AND GLUCOSE-6-PHOSPHATASE ACTIVITY OF HEPATOCYTES SEPARATED BY SEDIMENTATION. (Eng.) Tongiani, R.; Bibbiani, C.; Malvaldi, G. (Istituto di Patologia Generale, Universita degli Studi di Pisa, Scuola Medica, Via Roma 55, I-56100 Pisa, Italy). *Acta Histochem. (Jena)* 59(2):232-238; 1977.

Isolated rat hepatocytes were subfractionated by isopycnic or velocity sedimentation, and dry mass, triglycerides content, and glucose-6-phosphatase activity of different subpopulations were determined. The distribution of the cell dry masses and dry mass per average cell were similar in all the fractions separated by isopycnic sedimentation. Velocity sedimentation allowed a satisfactory separation of cells of different dry mass. Triglyceride content and glucose-6-phosphatase activity of cells of the different fractions obtained by isopycnic sedimentation showed no statistically significant differences. Subpopulations fractionated by velocity sedimentation differed in both triglyceride content and glucose-6-phosphatase activity, which were substantially parallel to the cell dry mass. Isopycnic sedimentation is not suitable for subfractionation of hepatocytes on the basis of their dry weight; this result can be achieved by velocity sedimentation.

0929 A COMPARISON OF EFFECT OF INSULIN ON HEPATIC METABOLITES, GLUCONEOGENESIS, AND KETOGENESIS. (Eng.) McDaniel, H. G. (Veterans Admin. Hosp., Birmingham, AL 35206). *Am. J. Physiol.* 233(1):E13-E18; 1977.

The effect of insulin on hepatic metabolites was studied in tryptophan-treated rats, and this effect was correlated with insulin-induced suppression of fatty acid oxidation and conversion of labeled lactate to glucose. In 12-hr fasted rats given tryptophan (25 mg/100 g, i.p.), insulin (10 U/kg, i.m.) decreased the hepatic content of alanine and the three precursors of oxalacetate (malate, citrate, and aspartate) while elevating hepatic pyruvate. These changes are consistent with suppression of the pyruvate carboxylase step. Animals fasted for 24 hr lose the effect on oxalacetate precursors, and this correlated with a loss of suppression of hepatic ketones. The decrease in hepatic alanine and oxalacetate precursors was more sensitive than the blood sugar. However, the conversion of labeled lactate to glucose was not inhibited by insulin in 12-hr fasted animals. (+)-Decanoylcarnitine also produced a decrease in oxalacetate precursors comparable to insulin and a lowering of the blood sugar. However, in fasted animals not given tryptophan it did not alter the blood sugar. Therefore, in tryptophan-treated animals alterations of fatty acid oxidation by insulin or (+)-decanoylcarnitine produce a fall in oxalacetate precursors consistent with inhibition of pyruvate carboxylase; however, this does not equate with overall suppression of gluconeogenesis by either of these agents in the absence of tryptophan.

0930 RESTORATION OF GLYCOGENESIS IN HEPATOCYTES FROM STARVED RATS. (Eng.) Geelen, M. J. H.; Pruden, E. L.; Gibson, D. M. (Lab. Veterinary Biochemistry, State Univ. Utrecht, Utrecht, Netherlands). *Life Sci.* 20(6):1027-1034; 1977.

Hepatocytes from rats starved for 48 hr were studied to define conditions that permit glycogen synthesis following the provision of metabolic precursors *in vitro*. Hepatocytes from the starved rats synthesized glycogen only when incubated in the presence of both glucose and glucogenic precursors (combinations of alanine, glycerol, pyruvate, lactate, or fructose). Unlabeled glucose increased the net flux of [U-^{14}C]alanine or [U-^{14}C]lactate into glycogen over 30-fold. Unlabeled lactate and alanine facilitated the incorporation of [U-^{14}C]-

glucose into glycogen, even though the basal level with glucose alone was relatively high, as compared to basal glucose incorporation. In systems containing no added endocrines, glucose dampened glycogen phosphorylase activity in a cyclic AMP-independent fashion. Fructose was unable to mimic the effects of glucose on glycogen deposition and on glycogen phosphorylase activity. The results suggest that with these liver cell preparations, it is possible to accumulate glycogen and to explore the early changes that ensue in the transition from the "glucogenic" state of liver metabolism in starvation to the "lipogenic" state in refeeding.

0931 THE EFFECTS OF EXPERIMENTAL INFLAMMATION ON ADAPTIVE SYNTHESIS OF RAT LIVER FATTY ACID SYNTHETASE. (Eng.) Shutler, G. G.; Langstaff, J. M.; Jamieson, J. C.; Burton, D. N. (Dept. Microbiology, Univ. Manitoba, Winnipeg, Manitoba R3T 2N2, Canada). *Arch. Biochem. Biophys.* 183(2):710-717; 1977.

The effects of experimental inflammation, induced by the s.c. injection of oil of turpentine (0.5 ml/100 g) on adaptive synthesis of liver fatty acid synthetase were investigated in male rats. Liver levels of α_1-acid glycoprotein, an "acute-phase" protein known to be synthesized at an accelerated rate as a result of inflammation, were also measured. The increase in fatty acid synthetase activity in livers of rats that were starved and then fed a fat-free diet was suppressed to an extent dependent on the periods between fat-free feeding and inflammation and inflammation and sacrifice. Inflammation induced 2 hr after refeeding gave complete suppression, whereas inflammation after 10 hr of fat-free feeding had no suppressive effect. When induced 2.5 or 7.5 hr after refeeding, inflammation led to partial suppression of the increase in fatty acid synthetase activity. The increase in liver α_1-acid glycoprotein levels characteristic of inflammation was reduced in animals inflamed 7.5 or 10 hr after fat-free feeding, but was unaffected when inflammation was induced 2.5 hr after refeeding. The ratio of free to membrane-bound polyribosomes in the liver increased from 0.77 in rats that were neither starved nor fed a fat-free diet to 3.31 in rats that were starved and then fed a fat-free diet for 15 hr. When inflammation was induced 2.5 hr after refeeding, the ratio increased to only 1.74 after 15 hr of refeeding. Inflammation resulted in a marked reduction in the level of glycogen in the liver, regardless of the time of induction of inflammation and the dietary status of the animal. The findings may be explained by changes in the activities of enzymes of glycogen metabolism, which lead to enhanced glycogenolysis in response to inflammation.

0932 EFFECTS OF OLEATE, PALMITATE, AND OCTANOATE ON GLUCONEOGENESIS IN ISOLATED RABBIT LIVER CELLS. (Eng.) Zaleski, J.; Bryla, J. (Inst. Biochemistry, Univ. Warsaw, 02-089 Warsaw, Poland). *Arch. Biochem. Biophys.* 183(2):553-562; 1977.

The effect of oleate, palmitate, and octanoate on glucose formation was studied in rabbit hepatocytes with lactate or pyruvate as substrate. Octanoate was much more quickly oxidized and utilized for ketone body production than were oleate and palmitate. Among the fatty acids studied, only octanoate resulted in a marked increase of the 3-hydroxybutyrate/acetoacetate ratio. Each of the fatty acids stimulated glucose synthesis from pyruvate. The enhancement of gluconeogenesis by long-chain fatty acids was abolished after the addition of ammonia. Examination of the "crossover" plot revealed that the stimulatory effect of fatty acids was due to (a) a stimulation of pyruvate carboxylation, (b) a provision of reducing equivalents for glyceraldehyde phosphate dehydrogenase, and (c) an acceleration of flux through hexose diphosphatase. Moreover, palmitate and oleate resulted in an increased generation of mitochondrial phosphoenolpyruvate, while in the presence of octanoate, the activity of mitochondrial phosphoenolpyruvate carboxykinase was diminished. When lactate was used as the glucose precursor, palmitate and oleate increased glucose production by about 50% but did not affect the contribution of mitochondrial phosphoenolpyruvate carboxykinase to gluconeogenesis. In spite of the stimulation of both pyruvate carboxylase and hexose diphosphatase, as judged from the crossover plot, the addition of octanoate resulted in a marked inhibition of both glucose formation and mitochondrial generation of phosphoenolpyruvate. The inhibitory effect of octanoate was reversed by ammonia. The results indicate that fatty acids and ammonia are potent regulatory factors of both the rate of glucose formation and the contribution of mitochondrial phosphenolpyruvate carboxykinase to gluconeogenesis in hepatocytes of the fasted rabbit.

0933 ACTION OF COBALT CHLORIDE ON THE BIOSYNTHESIS, DEGRADATION, AND UTILIZATION OF HEME IN FETAL RAT LIVER. (Eng.) Woods, J. S.; Carver, G. T. (Natl. Inst. Environmental Health Sciences, P.O. Box 12233, Research Triangle Park, NC 27709). *Drug Metab. Dispos.* 5(5):487-492; 1977.

The effects of cobalt chloride on heme biosynthesis and degradation and on the utilization of heme in the synthesis of various hemoproteins in fetal rat liver were investigated. $CoCl_2$ (50 mg/kg) administered to 18-day pregnant rats resulted in inhibition of the heme-biosynthetic pathway enzymes, δ-aminolevulinic acid (ALA) synthetase, and ferrochelatase, and increased the activity of microsomal heme oxygenase in fetal liver. Concomitantly, a 75-95% reduction in the rate of ^{3}H-ALA incorporation into fetal mitochondrial hemes was observed, and a 50% depletion of total microsomal heme levels occurred. Treatment of pregnant rats with $CoCl_2$ on the 18th day of gestation also resulted in a 35-40% reduction in the activities of cytochrome oxidase and aminopyrine demethylase measured in neonatal liver at 1 week after birth; deficiencies in both mitochondrial and microsomal hemoprotein function persisted for several weeks postnatally. Thus, $CoCl_2$ substantially alters hepatic heme metabolism in

fetal liver. Utilization of agents such as $CoCl_2$ as pharmacological tools may permit a clearer understanding of the gestational role of heme in the perinatal development of hepatic hemoprotein function.

0934 LIPOGENESIS IN HEPATOCYTES OF GENETICALLY OBESE RATS. (Eng.) Bloxham, D. P.; Fitzsimons, J. T. R.; York, D. A. (Dept. Physiology and Biochemistry, Univ. Southampton, Southampton SO9 3TU, England). *Horm. Metab. Res.* 9(4):304-309; 1977.

Lipogenesis from (U-^{14}C)lactate, (U-^{14}C)glucose, and tritiated water ($^{3}H_2O$) was investigated in hepatocytes obtained from both genetically obese rats and their lean littermates by the technique of nonrecirculating perfusion by gravity. Analysis of the incorporation of ^{14}C (microatoms of C/100 mg protein) from (U-^{14}C)lactate into various lipid fractions revealed a clear difference in the lipogenic capacity of hepatocytes from obese and lean rats on a standard chow diet. For hepatocytes from obese rats, the major product was triglyceride fatty acids (1.95 ± 0.11 versus 0.18 ± 0.01 microatoms of C/100 mg protein for lean hepatocytes, $p<0.001$), whereas for hepatocytes from lean rats the main product was glyceride-glycerol (3.48 ± 0.23 versus 0.61 ± 0.07 microatoms C/100 mg protein for obese hepatocytes, $p<0.001$) when these values were measured after 60 min of incubation with lactate. Sterol synthesis at this time was significantly higher ($p<0.001$) in hepatocytes from obese rats compared with lean rats (0.33 ± 0.04 versus 0.05 ± 0.01 microatoms of C/100 mg protein, resp.). There was virtually no difference in glucose incorporation into fatty acids in hepatocytes from either lean or obese rats, demonstrating that enhancement in hepatic fatty acid synthesis in the obese syndrome is related to increased activity of enzymes involved in lactate utilization rather than in enhanced conversion of glucose to lactate. Fatty acid synthesis increased in both lean and obese rats on a fat-free diet and fell markedly in both lean and obese rats when they were starved. Fatty acid synthesis in hepatocytes from obese rats was more resistant to starvation than in lean animals since fatty acid synthesis declined only to a rate of 0.23 microatoms C/100 mg protein/30 min in obese rats, whereas fatty acid synthesis was negligible in starved lean rats. When obese rats were changed from a chow diet to a fat free diet, lactate incorporation changed from being about equally divided between fatty acids and glyceride-glycerol to a 66% incorporation for fatty acids and a 19.3% incorporation for glyceride-glycerol. Following starvation in lean and obese rats, glyceride-glycerol represented the major point of lactate incorporation (54% for obese and 91% for lean). Studies involving the simultaneous incubation of hepatocytes with $^{3}H_2O$ and (U-^{14}C)-labeled substrates indicated that lactate is a major source of carbon for fatty acid synthesis at low concentrations and possibly the only source at high concentrations.

0935 PLASMA LIPOPROTEIN CHANGES IN EXPERIMENTAL CHOLESTASIS IN THE DOG. (Eng.) Äänelsson, B.; Ekman, R.; Johansson, B. G.; Petersson, B. G. (Univ. Hosp., Lund, Sweden). *Clin. Chim. Acta* 80(1):157-170; 1977.

Using zonal ultracentrifugation, gel electrophoresis, and Sephadex gel chromatography, plasma lipoproteins were studied after 1 week of complete biliary obstruction in the dog. The common bile duct was ligated and transected in eight dogs, and two dogs were sham-operated. A considerable increase in low density lipoprotein (LDL) was found 1 week after operation, with a zonal ultracentrifugal pattern that was more complex than that found before operation. The lipid composition was characterized by an increase of phospholipids and a decrease of triglycerides in cholestatic LDL fractions. None of the fractions displayed the inverse cholesterol-cholesterol ester ratio characteristic for human LP-X. The apoprotein composition of cholestatic LDL was also markedly changed. Apoprotein A I, already present in the normal LDL_2 subclass, increased, and two apoproteins with apparent molecular weights of 35,000–40,000 appeared in considerable amounts. The amount of high density lipoprotein (HDL) was decreased during cholestasis without any appreciable changes in composition. The results support the hypothesis that lipoprotein changes in cholestasis are related to an abnormal transformation of HDL particles.

0936 STUDIES ON RAT LIVER PLASMA MEMBRANE: ALTERED PROTEIN AND PHOSPHOLIPID METABOLISM AFTER INJECTION OF D-GALACTOSAMINE. (Eng.) Bachmann, W.; Harms, E.; Hassels, B.; Henninger, H.; Reutter, W. (III Medizinische Abteilung des Krankenhauses Schwabing und Forschergruppe Diabetes, D-8000 Munchen, Kolner Platz 1, W. Germany). *Biochem. J.* 166(3):455-462; 1977.

The metabolism of protein and phospholipid in isolated rat liver plasma membranes was investigated 3 and 6 hr after the injection of D-galactosamine (375 mg/kg, i.p.) *in vivo*. During this time, all the biochemical and morphological alterations associated with hepatitis developed. After the injection of D-galactosamine the concentration of sphingomyelin in the plasma membrane decreased to below 60% of the control values. The activity of 5'-nucleotidase (EC3.1.3.5), which has been purified as a sphingomyelin-protein complex, decreased in the total homogenate as well as in the plasma-membrane fraction of livers of rats treated with galactosamine, to about 60% of the control values. Protein synthesis, as measured by the incorporation of [^{14}C]leucine into plasma membranes, was decreased to 45% of that of the controls. However, only small differences were observed in the amino acid composition of the plasma membrane after D-galactosamine treatment. The protein composition of the plasma membranes was determined by sodium dodecyl sulfate/polyacrylamide-gel electrophoresis. The results showed a change from low- to high-molecular weight proteins after the injection of galactosamine. These results demonstrate different metabolic processes of the plasma membrane altered during the induction of galactosamine hepatitis.

0937 THE ACTIVITY OF SILYBIN ON PHOSPHOLIPID METABOLISM OF NORMAL AND FATTY LIVER IN VIVO. (Eng.) Castigli, E.; Montanini, I.; Roberti, R.; Porcellati, G. (Inst. Biochemistry, Univ. Perugia, 06100 Perugia, Italy). *Pharmacol. Res. Commun.* 9(1):59-69; 1977.

The mechanism by which silybin corrects ethanol- and ethionine-induced changes in phospholipid metabolism was investigated in the livers of female Wistar rats treated with a single dose of ethanol (0.6 g/100 g, p.o.) or with DL-ethionine (2 ml/150 g at 1, 8, 24, 32, and 48 hr, s.c.). Ethanol significantly increased the incorporation rate of labeled orthophosphate (10 μCi, i.p.) into liver phosphatidylcholine (Ptd-Cho, 14.7 ± 1.62 counts per minute [cpm]/μg phospholipid [P] in control experiments versus 20.9 ± 2.14 cpm/μg P, $p<0.001$), but did not affect orthophosphate incorporation into liver phosphatidylethanolamine (Ptd-Etn). Ethionine treatment inhibited the incorporation of labeled phosphate into both Ptd-Cho (16.1 ± 2.11 cpm/μg P versus 6.7 ± 0.86 cpm/mg P, $p<0.001$) and Ptd-Etn (11.2 ± 1.32 cpm/μg P versus 5.4 ± 0.61 cpm/μg P, $p<0.001$). The administration of silybin hemisuccinate sodium salt (150.6 mg/kg/day, i.v., corresponding to 100 mg/kg/day silybin base) for 5 days before the administration of ethionine or ethanol resulted in a Ptd-Cho level in the ethanol-treated rats nearly as low as the control level (15.2 ± 2.16 cpm/μg P). This result was probably due to the fact that the administration of silybin alone slightly decreased the rate of phosphate incorporation into lipid. Silybin administration significantly increased the rate of labeled phosphate into both Ptd-Cho (10.1 ± 1.12 cpm/μg P) and Ptd-Etn (7.5 ± 0.89 cpm/μg P) in the ethionine-treated rats, although not to control levels. In the untreated rat liver, silybin treatment did not inhibit phospholipid synthesis from choline, ethanolamine, or phosphate. The results suggest that the effect of silybin on the normal liver is different from that in ethanol- or ethionine-induced fatty liver, which is probably a result of a change in membrane structure in the fatty liver.

0938 THE INTERACTION OF HEPATOCYTE PLASMA MEMBRANES WITH AN AZIDE DERIVATIVE OF PROCAINE. (Eng.) Levy, D.; Glover, E.; Cheng, S. (Univ. Southern California, Sch. Medicine, Los Angeles, CA 90033). *Biochim. Biophys. Acta* 469(2):194-201; 1977.

The binding sites of procaine were characterized in rat liver plasma membranes. A photoreactive derivative of procaine, *p*-azidobenzoyldiethylaminoethanol hydrochloride, was synthesized and used as a site-directed probe to label hepatocyte plasma membranes. The procaine derivative was shown to have membrane binding and Ca^{2+} displacement characteristics quite similar to that of procaine. Photolysis of the derivative in the presence of hepatocyte plasma membranes resulted in the covalent incorporation of the probe into both the protein and lipid fractions. Analysis of the labeled membranes by sodium dodecyl sulfate-polyacrylamide gel electrophoresis indicated that one membrane protein was significantly labeled with a molecular weight of 21,400 in addition to membrane lipids. Both binding and labeling could be inhibited in the presence of an excess of procaine. The labeled membrane components may be involved in the binding of Ca^{2+} to the membrane system.

0939 THE METABOLISM OF CHYLOMICRON REMNANT BY ISOLATED PERFUSED RAT LIVER. (Eng.) Cooper, A. D. (Stanford Univ. Sch. Medicine, Stanford, CA 94305). *Biochim. Biophys. Acta* 488(3): 464-474; 1977.

The isolated perfused rat liver was used to study the hepatic metabolism of chylomicron remnants and to assess the hypothesis that they are the lipoprotein particles of dietary origin that can exert physiologic regulation of hepatic cholesterol synthesis. When whole chylomicrons containing radiolabeled lipids were added to the perfusate of an isolated liver, virtually no cholesterol ester or triacylglycerol was removed during the first hour, while a small amount of free cholesterol disappeared. After 3 hr, 40% of the cholesterol ester was removed by the liver. When chylomicron remnants, prepared by incubation of chylomicrons with postheparin plasma and containing the same amount of cholesterol, were added to the perfusate, 76 ± 7% of the cholesterol ester was removed in the first hour. Moreover, free cholesterol and triacylcerol disappeared from the perfusate at the same rate as the cholesterol ester, and, during the early phase of the perfusion, the total perfusate cholesterol content declined by the same amount as the radioactive cholesterol content. These results suggest a specific removal of the intact lipoprotein particle. Of the cholesterol ester removed, 42% was hydrolyzed to free cholesterol after 3 hr. When whole chylomicrons, containing the same amount of cholesterol, were injected into rats *in vivo*, 71 ± 4% of the cholesterol ester was removed by the liver in 3 hr, and 53% of this was hydrolyzed. The activity of 3-hydroxy-3-methylglutaryl-coenzyme A reductase, the rate-limiting enzyme of cholesterol synthesis, increased during liver perfusion with plasma-free or chylomicron-containing perfusate while addition of chylomicron remnants to the perfusate significantly diminished the effect. It is concluded that the chylomicron remnant is the lipoprotein of alimentary origin that regulates hepatic cholesterol synthesis, and that its metabolism by isolated liver appears to reflect the hepatic component of chylomicron metabolism *in vivo*.

0940 BILE CANALICULAR MEMBRANE PATHOLOGY IN CYTOCHALASIN B-INDUCED CHOLESTASIS. (Eng.) Oda, M.; Phillips, M. J. (Dept. Pathology, Univ. Toronto, 100 College St., Toronto, Ontario, Canada). *Lab. Invest.* 37(4):350-356; 1977.

The mechanism of cytochalasin B-induced intrahepatic cholestasis was examined in both isolated Wistar rat hepatocytes and *in vivo* infused liver using electron cytochemical techniques. Markers were used for three canalicular membrane components,

namely ruthenium red for the glycoprotein-rich surface coat, the Mg^{2+}-ATPase reaction as an example of a membrane-bound protein, and uranyl acetate *en bloc* and ruthenium red staining for the canalicular membrane-associated microfilaments. In rat liver infused *in vivo* with cytochalasin B, reduction in bile flow correlated with bile canalicular dilation, loss of the ruthenium red-positive surface coat from the canalicular membrane, and loss of demonstrable Mg^{2+}-ATPase activity. In addition, structural alterations in microfilaments with widening of the ectoplasmic zone were noted. In isolated liver cells, identical changes were found. Bile canaliculi isolated from the *in vivo* cytochalasin B-infused rat liver lacked their normal investment of microfilaments. Detachment of the filaments from the bile canalicular membrane may be involved in the mechanism of cytochalasin B-induced cholestasis.

0941 THE EFFECT OF DIETARY PROTEIN DEPRIVATION ON PROTEIN SYNTHESIS IN THE ISOLATED LIVER PARENCHYMAL CELL. (Eng.) Grant, A. G.; Hoffenberg, R. (St. George's Hosp. Medical Sch., Cranmer Terrace, Tooting, London SW17 ORE, England). *Br. J. Nutr.* 38(2):255-260; 1977.

Protein synthesis was compared in hepatic parenchymal cells from normal rats and from rats that had been deprived of dietary protein for 3 weeks. The cells from protein-deprived rats were two-thirds the diameter of those derived from livers of normal animals, and they consumed oxygen at a rate of 16.2 ± 2.7 µl/hr/10^6 viable cells, half the normal value. Albumin and transferrin were synthesized at rates of 0.94 ± 0.12 and 0.60 ± 0.07 µg/hr/10^6 viable cells, resp., and urea at a rate of 0.77 ± 0.12 µg/hr/10^6 cells from protein-deprived rats, which represents a 25-50% decrease in the rates of synthesis measured in cells isolated from normal livers. The 50% decrease in O_2 consumption in cells from protein-deprived rats is compatible with their smaller diameter and volume and suggests a decrease in either the size or number of mitochondria in each cell. The maintenance of cell viability and the inability of essential and nonessential amino acids to increase protein or urea synthesis in these cells suggests that the overall adaptation to protein deprivation is a general reduction in cell size rather than the withdrawal of cells from protein synthesis.

0942 CATABOLISM OF α-FETOPROTEIN IN CARBON TETRACHLORIDE-INJURED RATS. (Eng.) Watanabe, A.; Miyazaki, M. (Okayama Univ. Medical Sch., Okayama 700, Japan). *Acta Hepatogastroenterol.* 24(4):238-240; 1977.

The rate of alpha-fetoprotein (AFP) catabolism was determined in male Sprague-Dawley rats with carbon tetrachloride-induced liver injury and compared with that in normal rats. The rate of AFP catabolism in CCl_4-treated rats was estimated to be essentially the same as that found in control adult and young rats. The blockade of the reticuloendothelial system by carbon particles resulted in slightly shorter values for AFP half-life. The amount of AFP synthesized per day in CCl_4-treated rats was calculated to be nearly 10 times that in controls. Thus, the elevation of serum AFP concentrations in CCl_4-treated rats is derived from increased AFP synthesis by the injured liver.

0943 THE INFLUENCE OF INCREASED HEPATIC SEQUESTRATION AFTER SPLENECTOMY ON THE SURVIVAL AND OSMOTIC FRAGILITY OF RED CELLS IN RATS, WITH REFERENCE TO PROTEIN LEVELS IN DIETS. (Eng.) Hisaoka, F.; Shiraki, K.; Sagawa, S. (Sch. Medicine, Tokushima Univ., 3 Kuramotocho, 770 Tokushima, Japan). *J. Nutr. Sci. Vitaminol.* 23(3): 237-247; 1977.

The sequestration of RBC in rats by the liver following splenectomy was studied using an isologous ^{51}Cr-labeled population of either normal or *N*-ethylmaleimide (NEM) treated RBC. The spleen sequestered the damaged RBC selectively, while the liver compensated and overshot the sequestration for spleen after splenectomy. The sequestering response in liver increased gradually, reaching a maximum level 8 weeks after splenectomy and then declined toward the control level. These compensatory responses in liver were not observed in rats fed a low-protein diet, which indicated that the proliferative response imposed on liver by an extra work after splenectomy was not stimulated in the rats fed a low-protein diet. Splenectomy prolonged RBC survival and reduced the osmotic fragility of normal RBC, but the compensatory increase in sequestration of damaged RBC in the liver did not alter the survival and osmotic fragility of normal RBC of the rat. These findings indicate that the increased sequestration of reticuloendothelial cells in the liver is basically reparative and that it is impossible to compensate for the absence of the spleen because of an inability to duplicate certain anatomic features peculiar to the spleen.

0944 EFFECT OF GLUCOCORTICOID ON PROSTAGLANDIN PRODUCTION DURING SIMULATED SHOCK CONDITIONS IN THE PERFUSED CAT LIVER. (Eng.) Carlson, R. P.; Flynn, J. T.; Lefer, A. M. (Jefferson Medical Coll., Philadelphia, PA 19107). *Biochem. Pharmacol.* 26(16):1463-1468; 1977.

The production of prostaglandins E_2 and $F_{2\alpha}$ by isolated perfused cat livers was quantitated during separate simulated shock conditions (i.e., ischemia, acidosis, and hypoxia). Control livers showed little change in perfusate prostaglandin concentrations over a 150-min perfusion period. Neither ischemia nor acidosis produced significant increases in prostaglandin concentrations in the perfusate after 150 min of perfusion. Hypoxia resulted in a 4.5-fold increase in PGE_2 and a 3.5-fold increase in $PGF_{2\alpha}$ concentrations during this same period. Lactic acid dehydrogenase and cathepsin D activities were also elevated in the perfusate during hypoxia. The addition of methylprednisolone (10^{-3} M) to the perfusate significantly retarded the release of PGE_2, $PGF_{2\alpha}$, and cathepsin D into the per-

fusate during severe hypoxia, without significantly altering the increase in lactic acid dehydrogenase activity. The action of methylprednisolone on prostaglandin and lysosomal enzyme release in hypoxic livers may be the result of a membrane-stabilizing action of the glucocorticoid. One possible mechanism for the inhibition by glucocorticoids of the increased prostaglandin production during hypoxia is that endogenous arachidonic acid is stabilized within the cell membrane phospholipids, thus, less substrate is available for utilization by the prostaglandin synthetase system.

0945 THE EFFECT OF ANOXIA ON NITROGEN METABOLISM IN THE ISOLATED PERFUSED RAT LIVER. (Eng.)
Parrilla, R.; Goodman, M. N.; Toews, C. J. (Dept. Metabolism, Inst. G. Maranon, C.S.I.C., Madrid, Spain). *Pfluegers Arch.* 369(2):167-175; 1977.

The roles of different substrates and of anoxia in hepatic alanine metabolism were studied to elucidate the metabolic situations that would cause a reversal of the normal alanine flux from the extra- to the intra-cellular hepatic spaces. The total nitrogen balance of the liver was accounted for by proteolysis; production of alanine, glutamine, urea, and ammonia; and uptake of pyruvate, alanine, or glutamine uptake. The rate of alanine production was a function of the hepatic alanine concentration when it was not added as a substrate. Anoxia increased hepatic alanine production, largely due to an increased rate of proteolysis (chiefly of glutamate). However, a decrease in the glutamate level in the liver could not account for the increased alanine, thus suggesting intramitochondrial synthesis of glutamate that is then transferred to the cytosol. The mitochondrial $(NAD^+)/(NADH)$ ratio calculated from the glutamate or β-hydroxybutyrate dehydrogenase reactions was the same in both control and hypoxic livers, suggesting that the two enzymes are in equilibrium with the mitochondrial nicotinamide nucleotide pool. Anoxia had marked effects on nitrogen balance in the liver. When pyruvate was the substrate, the total hepatic nitrogen production increased, with most of the increase accounted for by alanine production. It is concluded that pyruvate availability is the critical factor in determining the rate of alanine production under all conditions studied and that increased proteolysis is the significant factor for increased hepatic nitrogen production during anoxia.

0946 DIFFERENTIAL ACTION OF PROGESTERONES ON HEPATIC MICROSOMAL ACTIVITIES IN THE RAT. (Eng.)
Feuer, G.; Kardish, R.; Farkas, R. (Banting Inst., Toronto, Ontario M5G 1L5, Canada). *Biochem. Pharmacol.* 26(16):1495-1499; 1977.

To test the hypothesis that changes in normal steroid balance could influence the metabolism of drugs and the pattern of liver response, the effect of the administration of reduced derivatives of progesterone (5α-pregnane-3β-ol-20-one, 5β-pregnane-3α-ol-20-one, 5α-pregnane-3β,20β-diol, and 5β-pregnane-3α,20α-diol) on the microsomal hydroxylation of selected drugs and of progesterone was measured in rats. The s.c. administration of these steroids in seven daily doses (20 mg/kg) significantly reduced the activities of aminopyrine *N*-demethylase, coumarin 3-hydroxylase, and glucose 6-phosphatase in hepatic microsomes. These steroids slightly raised inosine diphosphatase activity. 16α-Hydroxyprogesterone and pregnenolone-16α-carbonitrile significantly increased drug metabolism and slightly elevated glucose 6-phosphatase. The contrasting action of the different progesterone derivatives was associated with changes in microsomal phospholipid synthesis. Pregnanolone and pregnanediol significantly decreased the *de novo* incorporation of [^{14}C-methyl]-L-methionine into microsomal phospholipids, mainly manifesting in phosphatidylcholine, phosphatidylethanolamine, and lysophosphatidylcholine fractions; reduced the activity of S-adenosyl-L-methionine:microsomal-phosphatidylethanolamine methyl transferase; and caused a reduction of total microsomal phosphatidylcholine:phosphatidylethanolamine ratio. In contrast, 16α-hydroxy-progesterone and pregnenolone-16α-carbonitrile increased the *de novo* synthesis of microsomal phospholipids, methyl transferase activity, and the ratio of total microsomal phosphatidylcholine: phosphatidylethanolamine. Treatment with reduced progesterone derivatives diminished microsomal progesterone hydroxylation in the 16α- and 6β-position and raised progesterone Δ^4-5α-dehydrogenase activity measured *in vitro*. 16α-Hydroxyprogesterone and pregnenolone-16α-carbonitrile elevated progesterone hydroxylation. Considering these opposite effects, it can be postulated that, in the rat, the induction of drug metabolizing activity of the hepatic endoplasmic reticulum might be controlled by a balance displayed in the synthesis and metabolism of various progesterone derivatives.

0947 BIOTRANSFORMATION OF DRUGS AND THEIR INTERACTION WITH BARBITURATES. (Ger.)
Remmer, H. (Institut fur Toxikologie, Universitat Tubingen, Wilhelmstr. 56, 7400 Tubingen, W. Germany). *Z. Gastroenterol.* 15(6):362-372; 1977.

Studies on drug metabolism by liver enzymes are reviewed. The formation of oxidized compounds is the first step of the enzymatic biotransformation of drugs in the liver, which gives rise to radical intermediates that may elicit toxic action if not removed immediately by consecutive enzymatic reactions. The problem of liver enzyme induction by prescription drugs is generally overemphasized; the therapeutic doses used are normally too low to evoke increased enzyme production. Only few therapeutic drugs are known to act as enzyme inducers, either because they have to be used in high doses (e.g., rifampicin) or because of their ability to accumulate during treatment (e.g., phenobarbital, phenytoin). The inducing action is dependent on the drug concentration in the liver and inversely related to the rate of the breakdown of the drug. Inhibition of drug metabolism seems to be more important in therapy: as exogenous and endogenous liposoluble compounds compete for the binding sites of the oxidizing and conjugating enzymes, the release of a drug from the enzyme site depends on

the concentration and affinity of the displacing agent. Only drugs administered in a high dose range are able to inhibit the metabolism of another drug administered in a lower dose. Such competitive inhibition of metabolism leads to an increase in the therapeutic effect, and possibly also the toxic effect, of the drug whose metabolism is inhibited. If a drug produces the same therapeutic effect and has a similar therapeutic index as another drug, but is administered in a lower dose, an interaction with the second drug is very unlikely. Such combinations of drugs should be preferably prescribed to prevent interactions.

0948 EFFECT OF EXPERIMENTAL HEPATIC INJURY ON IN VITRO DRUG-METABOLIZING ENZYME ACTIVITIES IN THE RAT. (Eng.) Willson, R. A.; Hart, F. E. (Harborview Medical Center, 325 9th Ave., Seattle, WA 98104). *Gastroenterology* 73(4):691-696; 1977.

The relationship between *in vitro* drug metabolism, liver necrosis, and liver function test results was studied in male Sprague-Dawley rats with acetaminophen-induced liver injury. Hepatic necrosis was induced in phenobarbital-pretreated rats by the i.p. administration of an acetaminophen-dimethyl sulfoxide mixture (0.3-1.5 g/kg). At lower doses, cytochrome P-450 concentration and the specific activities of aminopyrine demethylase and bilirubin glucuronyl transferase increased, whereas aniline hydroxylase activity was mildly decreased. In contrast, at higher doses, cytochrome P-450 concentration and the specific activities of all of the enzymes were decreased significantly ($p<0.01$), as compared with control rats. The diminished activities, however, were generally modest, not uniform for all enzymes assayed, and correlated poorly with histological necrosis and routine laboratory tests. When necrosis was severe, a prominent peak was detected at 420 nm, and this was associated with a more consequential decrease in all of the enzyme activities. When the total *in vitro* hepatic drug metabolizing enzyme capacity was calculated, only the cytochrome P-450 content and aminopyrine demethylase activity were significantly decreased at the 1.5-g/kg dose. It is concluded that, in acute liver disease, changes in the hepatic *in vitro* drug metabolizing enzyme capacity may not be closely related to cellular necrosis, *per se*, and the degree of change in enzyme activities varies from one enzyme system to another. These findings may explain, in part, the often inconsistent alterations in the disposition and elimination of drugs described in associated liver diseases.

0949 INTERFERENCE OF CCl_4 METABOLITES WITH SUBCELLULAR STRUCTURES. (Eng.) Dianzani, M. U.; Gabriel, L.; Gravela, E.; Paradisi, L. (Istituto di Patologia Generale, Corso Raffaello 30, Torino, Italy). *Panminerva Med.* 18(9/10):310-319; 1976.

The effect of aldehydes and ketoaldehydes on the amino acid incorporating activity of the postmitochondrial supernatant from rat liver was studied to determine whether these substances, which are produced during the peroxidation of unsaturated fatty acids, could be involved in carbon tetrachloride-induced liver injury. *In vitro* protein synthesis was inhibited by 10 mM malonyldialdehyde, 5 mM 2-ketopentanal, 1 mM 4-hydroxypentenal, and 0.25 mM malealdehyde. Thiol compounds protected against 4-hydroxypentenal- and malealdehyde-induced inhibition of protein synthesis, indicating that the inhibition is dependent upon a blocking of sulfhydryl groups. CCl_4 (50 mM) inhibited polyuridine-directed phenylalanine incorporation in the postmitochondrial supernatant to 54% of the control level. The degree of inhibition was not affected by NADPH, and antioxidants did not protect against the inhibi- 0.1 mM $CBrCl_3$. This inhibition was intensified by NADPH. Polyuridine-directed polyphenylalanine synthesis was more severly impaired than was endogenous messenger RNA-dependent synthesis.

0950 ADVERSE EFFECTS OF CHLORPROMAZINE METABOLITES ON ISOLATED HEPATOCYTES. (Eng.) Abernathy, C. O.; Lukacs, L.; Zimmerman, H. J. (Veterans Admin. Hosp., 50 Irving St., N.W., Washington, DC 20422). *Proc. Soc. Exp. Biol. Med.* 155(4):474-478; 1977.

Chlorpromazine (CPZ) and several of its metabolites were tested for *in vitro* cytotoxicity, as measured by the efflux of aspartate aminotransferase to the surrounding medium, toward isolated rat hepatocytes. Exposure of liver cells to CPZ (9×10^{-5} M) led to enzyme leakage. The demethylated metabolites, mono- and didesmethyl-CPZ, were three and six times, resp., more potent than CPZ. Hydroxylation of the tricyclic ring at the 7 or 8 position gave rise to compounds that were slightly less active than the parent compound, while oxidation of the sulfur atom resulted in inactive analogs. The presence of calcium in the medium had no apparent effect on the response of the hepatocytes to CPZ. The results are consistent with the hypothesis that certain drugs, which cause jaundice in a small percentage of patients, possess a slight intrinsic toxicity.

0951 COMPARATIVE TOXICITIES OF ENFLURANE, FLUROXENE AND NITROUS OXIDE AT SUB-ANAESTHETIC CONCENTRATIONS IN LABORATORY ANIMALS. (Eng.) Stevens, W. C.; Eger, E. I., II; White, A.; Biava, C. G.; Gibbons, R. D.; Shargel, R. (Univ. California Medical Center, 436S, San Francisco, CA 94143). *Can. Anaesth. Soc. J.* 24(4):479-490; 1977.

The toxicities of subanesthetic concentrations of fluroxene, enflurane, and nitrous oxide were compared in mice, rats, and guinea pigs, which were all young and in an active growth phase. Fluroxene (>0.001% of air delivered in a closed chamber) produced a greater mortality and decrement in weight gain than did enflurane and nitrous oxide, despite the administration of far lower concentrations. All species that received 0.2% enflurane gained less

weight than controls. Reduction of enflurane to 0.07% reduced the detrimental effect on all species. Five male mice receiving 0.2% enflurane died by day 5, and by day 17, nine male and six female mice receiving 0.07% died. No rats receiving enflurane died. One male and two female guinea pigs that received 0.07% enflurane died. Of the controls, only one guinea pig died. Mice treated with 20% nitrous oxide gained more weight than controls. Experimental and control rats had similar weight gain, but experimental guinea pigs gained less weight than controls. Of the experimental animals, only two guinea pigs died, whereas two mice and one guinea pig in the control group died. Control female mice for the 0.01% fluroxene group were significantly heavier than the experimental mice before anesthesia administration, and this difference increased significantly at 4 days. Experimental male mice receiving 0.003% fluroxene were significantly heavier than the control mice before anesthesia, but this difference was reversed in only 5 days. Fluroxene was lethal to mice at 0.003%, and was poorly tolerated by any species for even brief periods at 0.01%. Nitrous oxide (0.1 minimum alveolar concentration) resulted in an increased incidence of focal inflammatory liver changes in mice (occurring in 5/15 controls and 34/42 experimental animals). Renal tubule calcification occurred in 2 of 7 controls and 5 of 10 experimental guinea pigs. Histologic changes produced by the other two agents were inconsistent. This study confirms the toxicity of fluoroxene in laboratory animals.

0952 THE ROLE OF BILIARY EXCRETION IN THE HEPATOTOXICITY OF FUROSEMIDE IN THE MOUSE. (Eng.) Spitznagle, L. A.; Wirth, P. J.; Boobis, S. W.; Thorgeirsson, S. S.; Nelson, W. L. (Dept. Pharmaceutical Sciences, Univ. Washington, Seattle, WA 98195). *Toxicol. Appl. Pharmacol.* 39(2):283-294; 1977.

The distribution of furosemide in the whole body and its binding to plasma proteins were studied in adult male Swiss-Webster mice or Swiss albino mice to elucidate the importance of biliary excretion in the hepatotoxicity of furosemide. Whole-body autoradiography (after administration, i.v. or i.p., of ^{35}S-furosemide, 150-300 mg/kg) showed that large amounts of furosemide and its metabolites accumulated in the gastrointestinal tract and that the excretory process through the biliary tract proceeded by a saturable, dose-dependent mechanism. More than 30% of the furosemide was excreted in the bile after doses of 50 and 100 mg/kg, but considerably less was excreted at higher doses. BSP administration decreased the biliary excretion and increased hepatic necrosis of furosemide (necrosis index was 2.63 when BSP was given with furosemide as compared to 1.07 with furosemide alone). However, equilibration of BSP with serum did not affect the binding of furosemide (5-500 µg/ml) to serum proteins. Two classes of binding sites for furosemide, differing in their association constants (KA), were found in the serum: $K_A=7.5 \times 10^4$ and 1.1×10^3. Total metabolism of furosemide and its covalent binding to liver proteins increased with increasing doses of the drug and correlated with corresponding increases in hepatotoxicity. It is concluded that biliary excretion is of considerable importance in the elimination of potentially hepatotoxic doses of furosemide in the mouse and that blocking this pathway (saturation with furosemide or BSP) leads to the observed hepatic necrosis.

0953 α-NAPHTHYLISOTHIOCYANATE (ANIT) HEPATOTOXICITY AND IRREVERSIBLE BINDING TO RAT LIVER MICROSOMES. (Eng.) El-Hawari, A. M.; Plaa, G. L. (Faculte de Medecine, Universite de Montreal, Montreal, Quebec, Canada). *Biochem. Pharmacol.* 26(20):1857-1866; 1977.

Experiments were carried out to determine whether the irreversible binding of α-naphthylisothiocyanate (ANIT) to hepatic macromolecules is related to its hepatotoxic effect. The binding of a chemically reactive ANIT-derived product to rat liver microsomes was examined *in vitro*. When [^{3}H]ANIT or [^{14}C]ANIT was incubated with rat liver microsomes, radioactivity was bound irreversibly to proteins. This binding occurred in the absence (nonenzymic) and, to greater extent, in the presence (enzymic) of NADPH and oxygen. Enzymic binding was inhibited by carbon monoxide, β-diethylaminoethyl diphenylpropylacetate (SKF 525-A), and piperonyl butoxide, indicating that a cytochrome P-450-dependent, mixed-function oxidase mediated the binding. The enzymic binding was reduced by incubation with glutathione and other thiol-containing compounds, while incubation with β-naphthylisothiocyanate or phenylisothiocyanate inhibited both the enzymic and nonenzymic binding. Pretreatment of rats with phenobarbital or 3-methylcholanthrene enhanced ANIT toxicity *in vivo* and markedly increased the irreversible binding *in vitro*. Piperonyl butoxide, SKF 525-A, disulfiram, and cobaltous chloride reduced ANIT toxicity and its binding to microsomes *in vitro*. However, cycloheximide, which abolished ANIT toxicity *in vivo*, failed to affect the binding. Furthermore, 16-α-pregnenolone carbonitrile reduced ANIT toxicity *in vivo* but increased its enzymic binding. The results demonstrate an inconsistent correlation between toxicity of ANIT *in vivo* and its binding to microsomal proteins *in vitro* and indicate that binding *in vitro* in itself is not a valid index of ANIT-induced hyperbilirubinemia. Limited studies *in vivo* in which the irreversible binding to liver proteins was examined after p.o. ANIT administration indicated that the extent of binding after different pretreatments also did not parallel ANIT toxicity. These data cast doubt on the role of such binding in the pathogenesis of ANIT-induced cholestasis.

0954 MICROSOMAL HEXOBARBITAL METABOLIZING ENZYMES IN REGENERATING CHOLESTATIC RAT LIVER. (Eng.) Ahonen, J.; Aronsen, K. F. (Helsinki Univ. Central Hosp., Helsinki, Finland). *Eur. Surg. Res.* 9(4):264-269; 1977.

Microsomal changes were studied in regenerating cholestatic rat liver by measuring hexobarbital metabolizing capacity (HMC) *in vitro*. Total extrahepatic biliary obstruction was induced in male Sprague-Dawley rats by common duct ligation, and 3 days later a 70% hepatectomy was carried out. In sham-operated control rats, hepatectomy resulted in a clear reduction in HMC. On day 7 after hepatectomy, an increase in HMC was observed. In the duct-ligated group, the initial value of HMC was reduced as compared with that in the sham-operated animals. Liver resection caused a further reduction in the activity, but the HMC activity started to increase at the same time as that in the sham-operated group. The similarity of the pattern of microsomal regeneration in duct-ligated and sham-operated rats indicates that cholestasis per se does not interfere with the development of the microsomal hexobarbital metabolizing enzyme system during regeneration.

0955 INDUCTION OF LIVER LYSOSOMAL ENZYMES DURING THE AUTOPHAGIC PHASE FOLLOWING PHENOBARBITAL TREATMENT OF RAT. (Eng.) Glaumann, H.; Arborgh, B.; Lindeborg, T. (Karolinska Institutet, Box 6401, S-113 82 Stockholm, Sweden). *Virchows Arch. [Cell Pathol.]* 23(1):17-28; 1977.

To find a possible biochemical correlation to the increased occurrence of autophagy observed after phenobarbital treatment, hydrolytic (lysosomal) enzymes were analyzed in rats during the autophagic phase. Male rats received phenobarbital (8 mg/100 g) i.p. either as a single dose or once daily for 7 days. The effects of treatment on the lysosomal hydrolases, acid phosphatase, cathepsin D, and aryl sulfatase were analyzed at different intervals ranging from 1 to 15 days after seven injections, and from 1 to 48 hr after a single injection. In both cases, microsomal protein and NADPH-cytochrome c reductase were measured to ensure proper induction. After a single injection, a slight decrease in hydrolytic activities was observed. Repeated administration of phenobarbital gave rise to a marked decrease of lysosomal enzyme activities 1 day after cessation of treatment. This decrease was followed by a continuous increase in activity up to days 3 and 4. One or 2 weeks after treatment, enzyme activities declined to control values. The increase in activity of lysosomal hydrolytic enzymes was correlated with the onset of induced autophagy of endoplasmic reticulum membranes described as occurring in liver upon cessation of phenobarbital exposure. It is concluded that phenobarbital treatment *per se* decreases lysosomal enzyme activities, whereas the induced autophagy following cessation of exposure is associated with enhanced levels of lysosomal hydrolases in rat liver.

0956 CYCLIC EXCRETION OF HEPATITIS A VIRUS IN EXPERIMENTALLY INFECTED CHIMPANZEES: BIOPHYSICAL CHARACTERIZATION OF THE ASSOCIATED HAV PARTICLES. (Eng.) Bradley, D. W.; Gravelle, C. R.; Cook, E. H.; Fields, R. M.; Maynard, J. E. (Center for Disease Control, 4402 North Seventh St., Phoenix, AZ 85014). *J. Med. Virol.* 1(2): 133-138; 1977.

The excretion pattern of hepatitis A virus (HAV) particles was studied in two experimentally infected chimpanzees to examine its relation to SGPT activity. The two chimpanzees were infected with Phoenix antigen of HAV. Radioimmunoassay of daily 10% stool suspensions showed a cyclic excretion of virus particles on days 9-11, 14-15, and 20-21 post-inoculation and a maximum in SGPT activity on days 30-31. Immune electron microscopy of stool filtrates showed the presence of predominantly empty (electron-lucent) HAV particles on day 10, but predominantly complete (electron-dense) HAV particles on day 14, suggesting that virus particles with different biophysical properties might be found in stools collected at different times after inoculation of the animal. Isopycnic banding in CsCl gradients of stool suspensions (17%) prepared from 9-11, 14-15, and 17, 19, and 21 day stool pools showed multiple buoyant densities for the associated HAV particles. Hollow HAV particles found in the 9-11 day pool banded primarily at a buoyant density of 1.30 g/cm^3. HAV particles in the 14-15 day pool and in the 17, 19, and 21 day pool banded bimodally with antigen peaks at buoyant densities of 1.29 and 1.33 g/cm^3 (14-15 day) and 1.33 and 1.40 g/cm^3 (17, 19, and 32 day), resp. Replication of HAV in experimentally infected chimpanzees is accompanied by the cyclic excretion of HAV particles that exhibit varying buoyant densities in CsCl gradients.

0957 CHRONIC BILE DUCT CANNULATION OF RHESUS MACAQUES (Macaca mulatta) WITHOUT CAUSING BILIARY FISTULAS. (Eng.) Cloyd, G. G.; Hiles, R. A.; Caudill, D. (Miami Valley Lab., Procter & Gamble Co., Cincinnati, OH 45247). *Am. J. Vet. Res.* 38(10):1607-1610; 1977.

0958 THE EFFECT OF CHRONIC ALCOHOL INTAKE UPON THE HEPATIC MICROSOMAL CARCINOGEN-ACTIVATION SYSTEM. (Eng.) Capel, I. D.; Williams, D. C. (Marie Curie Memorial Foundation, The Chart, Oxted, Surrey RH8 OTL, England). *IRCS Med. Sci.: Cancer* 5(9):448; 1977.

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0961 FREE AMINO ACIDS IN PLASMA DURING EXPERIMENTAL INFECTION OF MICE WITH THE MHV-3 STRAIN OF MOUSE HEPATITIS VIRUS. (Eng.) Cacciatore, L.; De Marco, F.; Cerini, R.; De Ritis, F. (II Sch. Medicine, Univ. Naples, Cappella dei Cangiani, 80131 Naples, Italy). *J. Infect. Dis.* 136(4):475-481; 1977.

0962 EFFECT OF EXPERIMENTAL SCHISTOSOMIASIS MANSONI ON PLASMA AND ERYTHROCYTE LIPIDS AND ON PLASMA PHOSPHATIDYLCHOLINE-CHOLESTEROL ACYLTRANSFERASE ACTIVITY IN THE MOUSE. (Eng.) Owen, J. S.; Costa, J. C. M.; Carvalho, V.; Gillett, M. P. T. (Royal Free Hosp., Pond St., London NW3 2QG, England). *Biochem. Soc. Trans.* 5(5):1346-1348; 1977.

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0965 NON-ADH PATHWAY OF ALCOHOL METABOLISM: ITS INCREASE IN ACTIVITY AT HIGH ETHANOL CONCENTRATIONS AND AFTER CHRONIC CONSUMPTION [Abstract]. (Eng.) Salaspuro, M.; Lieber, C. S. (Bronx Veterans Admin. Hosp., Bronx, NY). *Gastroenterology* 73(5):1245; 1977.

0966 STUDIES ON THE DRUG-INDUCED ALLERGIC INTRAHEPATIC CHOLESTASIS: HEPATIC CHOLESTASIS INDUCED BY LYMPHOCYTE CULTURE SUPERNATANT [Abstract]. (Eng.) Mizoguchi, Y.; Monna, T. (Osaka City Univ. Medical Sch., Osaka, Japan). *Gastroenterol. Jpn.* 12(4):336; 1977.

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0968 AVOIDANCE OF BLEEDING IN HEPATIC CRYOSURGERY BY AN IMPROVEMENT IN THE TECHNIQUE [Abstract]. (Eng.) Dutta, P.; Gage, A. A. (Veterans Admin. Hosp., Buffalo, NY 14215). *Cryobiology* 14(6):687; 1977.

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0977 ENTEROHEPATIC CIRCULATION OF INDIVIDUAL BILE ACIDS IN MAN: FASTING SERUM CONCENTRATIONS IN PORTAL VEIN AND SYSTEMIC CIRCULATION [Abstract]. (Eng.) Ahlberg, J.; Angelin, B.; Bjorkhem, I.; Einarsson, K. (Dept. Medicine and Surgery, Serafimerlasarettet, Stockholm, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):3; 1977.

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0980 DISPOSITION OF 4-DIMETHYL ^{14}C-AMINOANTIPYRINE (AMINOPYRINE) IN THE RAT: ANALYSIS OF TISSUE, PLASMA, URINE AND BREATH [Abstract]. (Eng.) Willson, R. A.; Hart, F. E.; Hew, J. T. (Dept. Medicine, Univ. Washington, Seattle, WA). *Gastroenterology* 73(5):1254; 1977.

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1003 EFFECT OF MEDIUM pH ON FECAL BILE ACID DEGRADATION [Abstract]. (Eng.) Macdonald, I. A.; Singh, G.; Mahony, D. E.; Meier, C. E. (Dept. Medicine, Dalhousie Univ., Halifax, Nova Scotia, Canada). *Clin. Res.* 25(5):688A; 1977.

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See also, 0784, 0788, 0790, 0834, 0891, 1019, 1037, 1078, 1100, 1108, 1136, 1234, 1275, 1377, 1389, 1391, 1408, 1472, 1481, 1488, 1491, 1527, 1535, 1536.

SECRETION AND METABOLISM

Intestine

1007 EFFECT OF LUMENAL CONTENTS ON COLONIC CELL REPLACEMENT. (Eng.) Stragand, J. J.; Hagemann, R. F. (Allegheny General Hosp., Pittsburgh, PA 15212). *Am. J. Physiol.* 233(3): E208-E211; 1977.

The role of physical versus nutritional stimulation in the response of colonic cell proliferation to fasting and refeeding (marked depression and transient stimulation, resp.) was evaluated in HA/ICR mice via a nondestructive colonic ligation procedure. The mice were fasted for 24 hr to remove food from the intestinal lumen. The colon was then ligated at the ascending region, 2 cm from the ileocecal junction. The fast was continued for an additional 48 hr. The mice were then refed for 8 hr, injected with tritiated thymidine, and sacrificed. The side of the colon proximal to the ligation and in contact with the food showed a dramatic response to refeeding (425 ± 75% greater cell renewal than that in sham-operated, nonfasting controls), whereas the side distal to the ligation remained depressed at the fasting level. The magnitude of ligation-induced colonic distention was such that the diameter of the proximal region of fasted and ligated animals was 1.5 times that of the controls and 2.6 times that of the distal region. The distal region of a fasted and ligated animal did not differ significantly from that of a fasted-only animal. The differences between the proximal and distal regions were similar

in magnitude to that seen within 8 hr after refeeding; the colonic diameter was increased 2.1 times from the fasting level. Ligation produced a marked increase in the proliferative activity of the proximal colon within 48 hr, and the hyperproliferation occurred despite continued fasting stress. The region distal to the ligation remained depressed throughout. The results suggest that physical stimulation by lumenal factors is in part required to initiate the colonic hyperplasia seen after refeeding. Physical stimulation may be manifested through the lumenal distention produced by newly ingested food.

1008 DIETARY INFLUENCE ON COLONIC CELL RENEWAL. (Eng.) Stragand, J. J.; Hagemann, R. F. (Allegheny General Hosp., 320 East North Ave., Pittsburgh, PA 15212). *Am. J. Clin. Nutr.* 30(6): 918-923; 1977.

The role of specific dietary components in the control of the response seen in the colon after starvation and refeeding and in normal colonic cell renewal was investigated in mice. The colonic refeeding response was dependent upon the presence of sugar, amino acids, and minerals (in the form of salts) in the refed diet. In the nonfasting animal, normal colonic cell renewal also required dietary minerals. The absence of minerals in a maintenance diet resulted in a marked hypoproliferative state that was reversed by reintroducing the mineral component of the diet. The depressed proliferative rate observed with a salt-free diet was not the result of a caloric depletion as demonstrated by the maintenance of body weight throughout the experimental period. These findings suggest a potential role for specific dietary components in the control of the colonic cell proliferation and in maintenance of the steady state cell renewal system, and represent a tool by which intestinal proliferative activity may be manipulated.

1009 ILEAL MUCOSAL CYCLIC AMP AND Cl SECRETION: SEROSAL VS. MUCOSAL ADDITION OF CHOLERA TOXIN. (Eng.) Sheerin, H. E.; Field, M. (Harvard Medical Sch., Boston, MA 02215). *Am. J. Physiol.* 232(2):E210-E215; 1977.

The effect of adding cholera toxin to the mucosal (CT_m) and serosal (CT_s) sides of isolated rabbit small intestine on cyclic (c) AMP concentration and ion transport was studied to elucidate the relationship between cAMP and secretion. Preparations of 2-cm segments of the distal ileum of New Zealand white male rabbits, stripped of muscle, were mounted in chambers containing bicarbonate-Ringer solution (pH 7.4), glucose, and mannitol (10 μmol/ml each). The transmural potential difference, short circuit current (SCC), and electrical resistance were determined. Cholera toxin (50 μg) was added to either the mucosal or serosal reservoirs, and ion flux was measured with tracer doses of ^{22}Na and ^{36}Cl. The concentration of cAMP in the tissue was measured in a protein-kinase binding assay by adding theophylline (5 μmol/min) to the reservoirs 180 and 280 min after toxin addition; after 10 min, the tissues were placed in cold 5% trichloroacetic acid containing a tracer dose of [^{3}H]cAMP. Toxin addition to CT_m significantly increased the SCC over control values after 60 min; however, the SCC of CT_s tissues increased slowly but steadily until, 280 min after toxin addition, it was significantly greater than that of CT_m tissue. Conductance was decreased 25% by CT_m 120 min after toxin addition, but was not affected by CT_s. CT_m inhibited the SCC increase produced by theophylline to less than 20% of control values by 180 min after toxin addition, whereas CT_s inhibited the SCC response to theophylline to 41% of control values by 280 min after toxin was added. Conductance of CT_m and CT_s tissues, unlike control tissues, was not significantly decreased by theophylline added 280 min after toxin. CT_m decreased Na^+ flux in both directions and the Cl^- flux from the mucosal to the serosal side, producing net Cl^- secretion. CT_s changed the Cl^- flux slightly in the secretory direction. CT_m and CT_s increased cAMP equally, when measured 190 min after toxin and 10 min after theophylline administration. Addition of toxin to both sides produced an additive increase in cAMP. The results suggest that much of the toxin-stimulated cAMP content of the mucosa is not coupled to secretion and that active secretion is probably stimulated by cholera toxin added on the serosal side, although more slowly than after addition to the mucosal side.

1010 STIMULATION OF CDP-CHOLINE BIOSYNTHESIS BY ENANTIOMERIC LYSOPHOSPHATIDYLCHOLINES IN RAT INTESTINAL MUCOSA. (Eng.) O'Doherty, P. J. A.; Smith, N. B.; Kuksis, A. (Dept. Biochemistry, Univ. Wisconsin, Madison, WI 53706). *Arch. Biochem. Biophys.* 180(1):10-18; 1977.

The effects of several molecular species of 3-*sn*-lysophosphatidylcholine and of 1-*sn*-lysophosphatidylcholine on the activity of phosphorylcholine cytidyltransferase were investigated in rat intestinal cells. The phosphorylcholine cytidyltransferase activity of the cytosol fractions of rat intestinal crypt and villus cells was stimulated by the addition of 3-*sn*-lysophosphatidylcholine. A 15- to 20-fold increase in activity was seen following a prior removal of phospholipids with acetone and butanol. 3-*sn*-Lysophosphatidylcholines of C_{16} and C_{18} carbon fatty acids were three to four times more effective than those with fatty chains of shorter length. The 3-*sn*-phosphatidyl and 3-*sn*-lysophosphatidyl ethanolamine, inositol, and serine were ineffective. 3-*sn*-Lysophosphatidic acid and 3-*sn*-glycerophosphorylcholine did not stimulate the transferase activity, indicating that both fatty acyl and phosphorylcholine moieties of the 3-*sn*-lysophosphatidylcholine molecule were required for the observed effect. Addition of the enantiomeric 1-*sn*-lysophosphatidylcholine had an effect comparable to that of 3-*sn*-lysophosphatidylcholine on the phosphorylcholine cytidyltransferase activity when assayed in rat intestinal mucosa and in pigeon liver. It is suggested that the stimulatory effect of lysophosphatidylcholine may be related to a specific de-

tergent property dependent upon the peculiar balance of hydrophilic and hydrophobic components in the molecule.

1011 INTESTINAL BRUSH BORDER AND LYSOSOMAL ENZYMES AND IMMUNOGLOBULIN ABSORPTION IN THE NEWLY-BORN LAMB. (Eng.) Healy, P. J. (Veterinary Res. Station, Glenfield, New South Wales, 2167, Australia). *Aust. J. Exp. Biol. Med. Sci.* 55(3):327-337; 1977.

The activities of the brush border enzymes alkaline phosphatase, leucine aminopeptidase, and lactase and the lysosomal enzymes α-mannosidase and β-*N*-acetylglucosaminidase were studied in newborn lambs fed colostrum. The objective was to determine whether the release of intestinal alkaline phosphatase into the circulation of these lambs is a consequence of absorption of immunoglobulins. In fed lambs (0-36 hr after birth), there were differences in enzyme activities in intestine and the serum, e.g., both alkaline phosphatase and α-mannosidase activities increased in serum but at 9 hr after birth intestinal alkaline phosphatase activity was higher ($p<0.001$) and intestinal α-mannosidase activity was lower in lambs fed colostrum than in fasted lambs. In lambs fed colostrum, serum globulin concentration increased with time until 15 hr after birth (mean concentration at 15 hr, 59.8 ± 5.9 mg/ml). Feeding lipid and protein components of colostrum and bovine serum resulted in enzyme responses similar to those observed after feeding colostrum in lambs fed 0-3 hr after birth. The enzyme activities increased in mesenteric lymph collected from newly born lambs when immunoglobulins were being absorbed from the jejunum and ileum. The results are consistent with the hypothesis that proteins are absorbed by pinocytosis and then transported to the intracellular spaces of the serosal side of the cells.

1012 INDUCTION OF CHICK EMBRYONIC INTESTINAL DISACCHARIDASES BY HYDROCORTISONE AND SUCROSE IN THE ORGAN CULTURE SYSTEM. (Eng.) Yoshizawa, S.; Moriuchi, S.; Hosoya, N. (Sch. Health Sciences, Univ. Tokyo, 3-1, Hongo 7, Bunkyo-ku, Tokyo 113, Japan). *J. Nutr. Sci. Vitaminol.* 23(3):227-235; 1977.

The effect of hydrocortisone and sucrose on the development of intestinal disaccharidases was studied in the cultured chick intestine. When jejuna of 15- and 17-day-old embryos were cultured in the presence of hydrocortisone (10^{-8}-10^{-5} M), disaccharidase activity was significantly increased, compared with the control. However, hydrocortisone had no effect in 20-day embryonic intestines. On the other hand, the disaccharidase activity of cultured intestines from 20-day-old chick embryos was significantly stimulated by the addition of sucrose. The increase in disaccharidase activity induced by hydrocortisone administration in 17-day-old embryos *in vitro* was inhibited 60% by actinomycin D and 70-100% by cycloheximide. The activity induced by the administration of sucrose in 20-day-old embryos *in vitro* was sensitive to cycloheximide but insensitive to actinomycin D. The inhibition of hydrocortisone-evoked disaccharidase activity by the administration of an inhibitor of messenger RNA synthesis indicates that the hormonal action was mediated by an effect on messenger RNA synthesis. It appears that sucrose might act on the glycosylation of enzymes occurring in chick intestine at the 20-day stage.

1013 INFLUENCE OF THYROID ON THE DEVELOPMENT OF INTESTINAL HYDROLASES. (Spa.) Llorens, J. M. C. (Red Cross Hosp., Barcelona, Spain). *Rev. Iber. Endocrinol.* 23(135):276-287; 1976.

The activity and distribution in distinct intestinal segments of disaccharidases and the levels of thyroxine in the blood during the perinatal period were simultaneously investigated. Female Sprague-Dawley rats were mated, and their litters were reduced to eight pups per mother at birth. On designated days after birth, the pups were decapitated, exsanguinated, and the serum tested for thyroxine; their intestines were removed and cut into portions 2-10 cm away from the pylorus. The distribution of the different disaccharidases was homogeneous throughout the intestinal segments tested. Values for sucrase and maltase increased, whereas lactase levels decreased with increasing age of the animals. Maltase values were 55.8 ± 21 U/g at 1-5 days, 31 ± 21 U/g at 9-13 days, and 135 ± 50 U/g at 13-33 days. Lactase levels at the same age intervals were 66 ± 18.8 U/g, 28 ± 21.4 U/g, and 10.2 ± 12.9 U/g, resp. On the other hand, the thyroxine levels were practically undetectable at birth (<0.05 μg/100 ml) and rapidly increased to the adult value (3.8 μg/100 ml) at 22 days after birth. It is concluded that the thyroid gland plays an important role in the intestinal maturation of rats.

1014 LIPOLYTIC ACTIVITIES AGAINST TRIOCTANOIN AND MONOOLEIN IN RAT INTESTINAL MUCOSA. (Eng.) Negrel, R.; Serrero, G.; Ailhaud, G. (Centre de Biochimie, Parc Valrose, 06034 Nice, France). *Biochimie* 59(4):433-438; 1977.

The hydrolysis of trioctanoin and of monoolein by rat intestinal homogenates, catalyzed by the previously described "trioctanoin hydrolase" and "monoglyceride lipase," was studied. The hydrolysis rate of trioctanoin catalyzed by homogeneous intestinal glycerol-ester hydrolase was 0.1% of that of tributyrin, indicating that this enzyme does not account for all the activity observed against trioctanoin (0.17 U/mg against a calculated value of 0.005 U/mg) in the homogenate. Treatment with 0.1 mM diisopropylphosphofluoridate, a glycerol-ester hydrolase inhibitor, caused no inhibition of triolein hydrolysis and some inhibition of trioctanoin hydrolysis. Tributyrin and dioctanoin hydrolysis was inhibited incompletely, while *p*-nitrophenylacetate hydrolysis was completely inhibited by diisopropylphosphofluoridate. Pancreatic lipase is known to remain fully active after diisopropylphosphofluoridate treatment;

tributyrin, trioctanoin, and dioctanoin are hydrolyzed by both glycerol-ester hydrolase and pancreatic lipase, while *p*-nitrophenylacetate and triolein are only cleaved by glycerol-ester hydrolase and by lipase, resp. It is therefore concluded that contaminating pancreatic lipase, together with glycerol-ester hydrolase, is responsible for trioctanoin and monoolein hydrolysis. Only 12% of the trioctanoin hydrolase activity was recovered at pH 7.5 in the supernatant fraction of the homogenate. Alkaline treatment of the resulting pellet lead to a 70% recovery of the activity measured with either trioctanoin or triolein as substrates. Diethylaminoethane (DEAE)-cellulose chromatography at pH 7.5 showed that all octanoin hydrolase and triolein hydrolase activity was eluted with the breakthrough volume, whereas 90% of the tributyrin hydrolase activity was retained. Immunoprecipitation studies showed complete precipitation of the activity measured against tributyrin and trioctanoin as substrates. Identical equivalence points were found with the pH 8.5 supernatant, the cationic fraction, and the homogeneous rat pancreatic lipase using immunoserum against rat pancreatic juice. The results support the conclusion that trioctanoin and monoolein hydrolyses are due entirely to the action of intestinal glycerol-ester hydrolase and to contaminating pancreatic lipase bound to the intestinal cells.

1015 CHARACTERISTICS AND POSTNATAL DEVELOPMENT OF THE ACID LIPASE ACTIVITY OF THE RAT SMALL INTESTINE. (Eng.) Coates, P. M.; Brown, S. A.; Jumawan, J.; Koldovsky, O. (Children's Hosp. Philadelphia, Philadelphia, PA 19104). *Biochem. J.* 166(3):331-338; 1977.

For the first time, acid lipase was identified in the rat small intestine by using esters of 4-methylumbelliferone as substrates; its biochemical characteristics and changes in its activity in various sections of the small intestine during early postnatal development are described. Maximum activity towards the oleate ester was found at pH 4.0. In adult animals, the activity of acid lipase exhibited both latency and sedimentability, indicating a lysosomal localization. The activity of acid lipase was practically the same along the height of the villus, thus paralleling the distribution of acid β-galactosidase. In adult rats, the activity of acid lipase in proximal jejunum and mid-jejunum sections of the small intestine was practically the same and exceeded the activity in the ileum by a factor of 2. In suckling rats, the activity of the enzyme in the mid-jejunum exceeded that in the proximal jejunum and ileum by 2.5- and 1.5-fold, resp. During postnatal development, the acid lipase activity of the mid-jejunum showed a peak between days 10 and 15, at which time it exceeded the adult mid-jejunum activity by five- to sixfold. The results of the pH-dependency experiments, studies of the distribution of the enzyme activities, and latency studies suggest the presence of more than one lipolytic activity hydrolyzing esters of 4-methylumbelliferone.

1016 METABOLISM AND TRANSPORT OF GLUTAMINE AND GLUCOSE IN VASCULARLY PERFUSED SMALL INTESTINE RAT [sic]. (Eng.) Hanson, P. J.; Parsons, D. S. (Dept. Biochemistry, Univ. Oxford, South Parks Rd., Oxford OX1 3QU, England). *Biochem. J.* 166(3): 509-519; 1977.

The metabolism and transport of glutamine and glucose were investigated in a preparation of rat small intestine perfused through the vascular bed *in vitro* and *in situ*. With glucose (7.5 mM) or glutamine (4.5 mM) in the lumen, approximately 40% of the substrate taken up appeared to be unchanged on the vascular side. When glutamine (1.5 mM) was also added to the vascular perfusate, glutamine metabolism was increased, and glutamine was taken up from both the vascular bed and lumen. When glutamine was presented to the tissue from the lumen, there was greater conversion into alanine, and when glucose was presented to the intestine from the lumen, there was greater conversion into lactate. Deprivation of food and metabolic acidosis had no effect upon glutamine utilization by unit length of jejunum. In fed rats, glutamine utilization was 44% of glucose utilization, but in rats deprived of food, it was 112% of glucose utilization. Glucose utilization and lactate production were not significantly altered by the presence of glutamine in the vascular bed or lumen. With glucose only in the vascular perfusate, glucose utilization was the same in jejunum and ileum. Glutamine metabolism in the ileum was 28% lower than in the jejunum. Glutamine utilization was dependent on the concentration of glutamine in the vascular perfusate, but was not significantly affected by the absence of glucose.

1017 BIOSYNTHESIS OF BRUSH BORDER GLYCOPROTEINS BY HUMAN SMALL INTESTINAL MUCOSA IN ORGAN CULTURE. (Eng.) Hauri, H. P.; Kedinger, M.; Haffen, K.; Freiburghaus, A.; Grenier, J. F.; Hadorn, B. (Dept. Pediatrics, Univ. Bern, Freiburgstr. 23, CH-3010 Bern, Switzerland). *Biochim. Biophys. Acta* 467(3):327-339; 1977.

The synthesis of brush border glycoproteins by human small intestinal mucosa from patients with morbid obesity was studied in organ culture by measuring the incorporation of ^{14}C-glucosamine into brush border membranes and the release of labeled proteins into the culture medium. Purified brush border membrane proteins were analyzed on polyacrylamide-sodium dodecyl sulfate gels. Nonsoluble proteins from the culture media were isolated for analysis by ultracentrifugation. Total ^{14}C-glucosamine (5 μCi/ml) incorporation into glycoproteins was measured by long-term and pulse labeling; incorporation into brush border proteins by culturing explants in the presence of ^{14}C-glucosamine (10 or 20 μCi/ml) and periodically harvesting specimens for analysis. There were no significant differences between the protein patterns after gel electrophoresis of cultured and noncultured explants. Radioactivity present in the tissue plus medium increased linearly over a period of 48 hr. Between 24 and 48 hr, the

rate of incorporation into tissue leveled off, but release of labeled proteins into the medium increased. After 5 hr, label incorporation into isolated brush border membranes was found in peaks comigrating with markers of maltase-glucoamylase, lactase, sucrase-maltase, and enterokinase plus one unidentified peak. After 24 hr, a peak co-migrating with alkaline phosphatase activity appeared as well as two more unidentified peaks. Incorporation of label into the nonsoluble fraction of the media revealed similar protein patterns. These results indicated that the brush border membrane remains functionally stable in organ culture, providing a useful system for studying the biosynthesis and molecular nature of glycoproteins produced by patients with congenital brush border enzyme deficiencies.

1018 PLASMA SECRETIN CONCENTRATION IN ANAESTHETIZED PIGS AFTER INTRADUODENAL GLUCOSE, FAT, AMINOACIDS, OR MEALS WITH VARIOUS pH. (Eng.) Fahrenkrug, J.; Schaffalitzky de Muckadell, O. B.; Holst, J. J. (Dept. Clinical Chemistry, Bispebjerg Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12(3):273-279; 1977.

The concentration of immunoreactive secretin in portal blood and the secretion from the exocrine pancreas were measured during intraduodenal infusion of glucose, fat, amino acids, or meals with various pH in seven anesthetized Danish Landrace pigs. The solutions (50 ml each) were warmed to 37 C and infused for 1 min each in the following order after a 15-min basal period: 0.9% NaCl, 5.5% glucose, isotonic fat emulsion, isotonic amino acids, 0.1 M HCl, 9.0% NaCl, and 50% glucose. All solutions except HCl were adjusted to pH 7.0. The duodenum was drained 15 min after an infusion and the next test solution was infused 15 min after the draining (30 min after HCl). In another experiment, test meals composed of 1.5 g protein, 1.2 g fat, and 0.4 g carbohydrate was infused at several pH's (7.0, 5.0, 4.0, 3.0, 2.0, and 1.0) into the duodenum of four pigs. The levels of immunoreactive secretin in portal blood were not affected by any of the solutions except HCl, which caused the mean plasma secretin level to increase from 5.6 ± 2.7 to 201.2 ± 80.5 pM in 15 min. HCl increased the mean pancreatic flow rate from 0.51 ± 0.19 to 9.85 ± 2.33 ml/hr and the bicarbonate output from 52 ± 11 to 1,004 ± 290 µmol/hr. All solutions, except those of NaCl increased protein output significantly, and hypertonic glucose and HCl significantly increased amylase levels. The plasma secretin level was significantly increased only by the test meal at pH 1.0 (from 5.5 ± 2.8 to 115.0 ± 51.2 pM after 15 min). Pancreatic flow increased from 0.20 ± 0.08 to 6.25 ± 2.57 ml/hr. Amylase levels were increased after all meals. Several inconsistencies with the results of previous studies are discussed, as well as the possibilities that the amount of acid infused with each meal might have been too low and that anesthesia may have influenced the amount of pancreatic secretion. It is concluded that the physiological role of secretin could not be clearly defined by this study.

1019 LACK OF HEPATIC INACTIVATION OF CANINE VASOACTIVE INTESTINAL PEPTIDE. (Eng.) Strunz, U. T.; Walsh, J. H.; Bloom, S. R.; Thompson, M. R.; Grossman, M. I. (Veterans Admin. Wadsworth Hosp. Center, Los Angeles, CA 90073). *Gastroenterology* 73(4):768-771; 1977.

Hepatic inactivation of vasoactive intestinal peptide (VIP) was studied in conscious dogs. In three animals with gastric fistula, the portal vein and inferior vena cava were transposed, thus allowing access to the portal vein through a hind leg vein. To demonstrate that the liver's ability to inactivate peptides was intact in these dogs, each experiment with VIP was preceded by an intraportal pentagastrin infusion (4 µg/kg/hr), which failed to stimulate acid secretion. In the tests with VIP, a plateau of acid secretion was established by systemic i.v. infusion of pentagastrin (1 µg/kg/hr). While continuing infusion of pentagastrin, VIP (0.5, 1.0, or 2.0 µg/kg/hr) was given by portal or systemic infusion for 30 min. VIP produced a dose-dependent decrease in acid secretion ranging from about 30% inhibition with the low dose to about 60% inhibition with the high dose. The degree of inhibition produced by portal infusion of VIP did not differ significantly from that produced by systemic infusion. After infusion of 30 µg/kg/hr of VIP for 5 min, peak serum VIP levels by radioimmunoassay were 769 ± 63 pM after systemic and 717 ± 60 pM after portal administration, an insignificant difference. After stopping the infusion, the disappearance half-time of VIP was 3.1 ± 0.3 min, which did not differ significantly with the two routes of administration. It is concluded that inhibition of acid secretion and serum levels of VIP were not significantly different after portal or systemic infusions of VIP. Thus, no evidence of selective hepatic inactivation of VIP was found in dogs.

1020 RELEASE OF VASOACTIVE INTESTINAL POLYPEPTIDE (VIP) BY INTRADUODENAL STIMULI [Abstract]. (Eng.) Schaffalitzky de Muckadell, O. B.; Fahrenkrug, J.; Holst, J. J. (Bispebjerg Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):89; 1977.

1021 IONIC REQUIREMENTS FOR CAERULEIN-STIMULATED RELEASE OF ACETYLCHOLINE FROM MYENTERIC PLEXUS OF SMALL INTESTINE [Abstract]. (Eng.) Yau, W. M.; Nemeth, P. R. (Sch. Medicine, Southern Illinois Univ., Carbondale, IL 62901). *Physiologist* 20(4):103; 1977.

1022 THE EFFECT OF ATROPINE ON SECRETIN RELEASE AND PANCREATIC BICARBONATE SECRETION AFTER DUODENAL ACIDIFICATION IN MAN [Abstract]. (Eng.) Hanssen, L. E. (Ulleval Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):32; 1977.

1023 REDUCTION OF BILE ACID- AND FATTY ACID-INDUCED INTESTINAL SECRETION BY PROPRANOLOL AND METHYLPREDNISOLONE [Abstract]. (Eng.) Feinstein, J. D.; Bonorris, G. G.; Chung, A.; Coyne,

M. J.; Schoenfield, L. J. (Cedars Sinai Hosp., Los Angeles, CA). *Clin. Res.* 25(4):607A; 1977.

1024 HALF-LIFE, CLEARANCE, AND ENDOGENOUS SECRETION RATES OF CHOLECYSTOKININ-PANCREOZYMIN IN MAN [Abstract]. (Eng.) Harvey, R. F.; Oliver, J. M.; Read, A. E.; Ederle, A.; Vantini, I.; Scuro, L. A.; *et al.* (Frenchay Hosp., Bristol, England). *Gut* 18(11):A982; 1977.

1025 RISE OF SECRETIN AFTER ORAL FEEDING [Abstract]. (Eng.) Greenberg, G. R.; Hacki, W.; Domschke, W.; Domschke, S.; Mitznegg, P.; Demling, L.; Bloom, S. R. (Hammersmith Hosp., London, England). *Gut* 18(11):A981; 1977.

1026 INHIBITION OF CYCLIC AMP PRODUCTION IN ISOLATED COLON CELLS BY SOMATOSTATIN [Abstract]. (Eng.) Carter, R. F.; Bitar, K. N.; Zfass, A. M.; Makhlouf, G. M. (Medical Coll. Virginia, Richmond, VA). *Clin. Res.* 25(4):570A; 1977.

1027 POSTPRANDIAL DUODENAL FUNCTION IN MAN [Abstract]. (Eng.) Miller, L. J.; Malagelada, J. R.; Go, V. L. W. (Mayo Clinic, Rochester, MN). *Clin. Res.* 25(4):573A; 1977.

1028 VASOACTIVE INTESTINAL PEPTIDE (VIP): QUANTIFICATION IN THE MAJOR COMPONENTS OF THE HAMSTER INTESTINE [Abstract]. (Eng.) O'Dorisio, T. M.; Mekhjian, H. S.; Gaginella, T. S. (Dept. Medicine, Ohio State Univ., Columbus, OH). *Clin. Res.* 25(4):573A; 1977.

1029 POSSIBLE DUAL ROLE FOR VASOACTIVE INTESTINAL PEPTIDE AS GASTROINTESTINAL HORMONE AND NEUROTRANSMITTER SUBSTANCE. (Eng.) Bryant, M. G.; Bloom, S. R.; Polak, J. M.; *et al.* (Hammersmith Hosp., London, England). *Lancet* 1(7967):991-993; 1976.

1030 DEVELOPMENT OF BILE ACID TRANSPORT IN HUMAN INTESTINE [Abstract]. (Eng.) Vaupshas, V.; Vitullo, B. B.; Mackie, G.; Shaffer, E.; de Belle, R. C. (Dept. Pediatrics, McGill Univ., Montreal, Canada). *Gastroenterology* 73(5):1252; 1977.

1031 UREA, URIC ACID, AND CREATININE FLUXES THROUGH THE SMALL INTESTINE OF MAN [Abstract]. (Eng.) Chadwick, V. S.; Jones, J. D.; Debongnie, J. C.; Gaginella, T.; Phillips, S. F. (Mayo Clinic, Rochester, MN 55901). *Gut* 18(11):A944; 1977.

1032 RELEASE OF VIP BY DUODENAL ACIDIFICATION IN MAN [Abstract]. (Eng.) Bloom, S. R.; Mitchell, S. J.; Greenberg, G. R.; Christofides, N.; Domschke, W.; Domschke, S.; *et al.* (Hammersmith Hosp., London, England). *Gut* 18(11):A963; 1977.

1033 CORRELATION BETWEEN THE SUCRASE ACTIVITY IN VITRO AND THE RATES OF DIGESTION AND ABSORPTION OF SUCPOSE IN VIVO IN RAT SMALL INTESTINE. (Jpn.) Murakami, E.; Saito, M.; Suda, M. (Sch. Medicine, Ehime Univ., Shigenobu, Ehime, Japan). *J. Jpn. Soc. Food Nutr.* 30(1):1-5; 1977.

1034 THE BIOCHEMICAL HETEROGENEITY OF MITOCHONDRIA FROM MUCOSA OF RAT SMALL INTESTINE. (Rus.) Adrianov, N. V.; Spirichev, V. B.; Shuppe, N. G. (Inst. Nutrition, Acad. Medical Science USSR, Moscow, USSR). *Vopr. Med. Khim.* 23(3): 398-403; 1977.

See also, 0778, 0779, 0780, 0781, 0788, 0789, 0799, 0800, 0803, 0814, 0834, 0856, 0888, 0898, 0914, 0999, 1038, 1039, 1041, 1044, 1053, 1054, 1059, 1069, 1074, 1081, 1216, 1266, 1353, 1623, 1637, 1638, 1661.

GENERAL

1035 THE MECHANISM OF LINCOMYCIN-INDUCED DIARRHOEA. (Eng.) Hoff, S. G.; Schjonsby, H.; Andersen, K. J. (Univ. Clinic, Bergen, Norway). *Acta Med. Scand. (Suppl.)* 603:35-38; 1977.

To determine whether lincomycin-induced diarrhea is caused by bile acid malabsorption, metabolic studies were performed before and 7 days after treating rats p.o. with lincomycin (1 mg/ml drinking water). Following the treatment the mean fecal weight increased from 30.2 ± 3.8 g/72 hr to 65.5 ± 8.2 g/72 hr. The fecal fat excretion was unchanged, and the weight increase was mainly due to increased water content. The absorption rate of ^{14}C-taurocholic acid was measured in untreated rats and rats treated with lincomycin, using an *in vivo* perfusion technique. There was no significant difference in bile acid absorption rate measured at three different concentrations of bile acid (0.125, 0.500, 1.500 mM) in the perfusate. Although these results apparently exclude the hypothesis that lincomycin-induced diarrhea is caused by bile acid malabsorption, a back-wash ileitis might be present in this type of diarrhea and could explain bile acid malabsorption as a secondary phenomenon.

1036 PROTEIN-ENERGY MALNUTRITION IN INFANT NON-HUMAN PRIMATES (Macaca nemestrina). I.

CORRELATION OF BIOCHEMICAL CHANGES WITH FINE STRUCTURAL ALTERATIONS IN THE LIVER. (Eng.) Enwonwu, C. O.; Worthington, B. S.; Jacobson, K. L. (Center for Res. Oral Biology, Univ. Washington, Seattle, WA). *Br. J. Exp. Pathol.* 58(1):78-94; 1977.

Possible correlations between biochemical changes and fine structural alterations were investigated in the livers of malnourished infant monkeys. Male infant pigtail monkeys *(Macaca nemestrina)* aged 6-9 months and born in captivity were fed a restricted amount of either an adequate protein diet (20% casein) or a protein-deficient diet (2% casein). After 3-5 months of receiving the low protein-containing diet, ultrastructural evaluation of hepatic parenchymal cells revealed extensive fatty metamorphosis, marked fragmentation and vesiculation of the rough endoplasmic reticulum, some evidence of distention of the nuclear membrane, a variable degree of mitochondrial polymorphism and gigantism, increased intracytoplasmic accumulation of glycogen particles, and enhanced prominence of collagen fibers in the intercellular and perisinusoidal spaces. Biochemical studies indicated severe distortion in the plasma profile of free amino acids by week 10 of malnutrition, with a prominent decrease in valine/glycine ratio (9% in the malnourished animals compared to a ratio of 55-63% in the control group). While plasma concentration of glycine was elevated above baseline value throughout the period of malnutrition, the level of alanine showed an initial rise followed by a significant drop (-58% at week 20). Although showing wide individual variation, the plasma level of corticosteroid in general tended to increase with duration of protein malnutrition, an observation consistent with the accumulation of glycogen and enlargement of mitochondria in the hepatic cells.

1037 DECREASED CONJUGATION OF p-AMINOBENZOIC ACID IN THE ICTERIC NEW BORN GUNN RAT. (Eng.) Davis, D. R.; Yeary, R. A. (Dept. Veterinary Physiology, Ohio State Univ., Columbus, OH 43210). *Biochem. Pharmacol.* 26(6):535-539; 1977.

The metabolism of *p*-aminobenzoic acid in the icteric and nonicteric Gunn rat was studied. The urinary metabolites of *p*-aminobenzoic acid identified were the parent compound, *p*-aminohippuric acid, and *p*-aminobenzoic acid glucuronide. The icteric Gunn rat excreted significantly less *p*-aminobenzoic acid within 24 hr and also excreted significantly less of the compound as the glucuronide. In the newborn icteric rat, the major excretory product was the unchanged *p*-aminobenzoic acid, whereas in the adult most of the drug was excreted as the glycine conjugate. The data *in vivo* and *in vitro* indicated that the icteric newborn rat converts significantly less *p*-aminobenzoic acid to *p*-aminohippuric acid than does the nonicteric rat. The synthesis of *p*-aminohippuric acid was low in both genotypes at birth, but rapidly approached adult levels at 30 days of age. The increased toxicity of *p*-aminobenzoic acid in the 2-day-old icteric Gunn rat may be the result of a decreased *p*-aminobenzoic acid metabolism in these rats due to a deficiency in the activity of the enzyme uridine diphosphate-glucuronyltransferase and possibly glycine-*N*-acyltransferase.

1038 ANGIOGRAPHIC ASPECTS OF EXPERIMENTAL NONOCCLUSIVE INTESTINAL ISCHEMIC INJURY. (Eng.) Bookstein, J. J.; Goldberger, L.; Niwayama, G.; Naderi, M. J.; Brahme, F. J.; Jones, T. A. (Univ. California Medical Center, 225 West Dickinson St., San Diego, CA 92103). *Am. J. Roentgenol.* 128(6):923-930; 1977.

Hypovolemic and normovolemic hypotensive models for nonocclusive ischemic injury were developed in mongrel dogs to investigate the accuracy of angiography in diagnosing the presence and extent of ischemic injury and to evaluate the therapeutic, prophylactic, or diagnostic values of papaverine as a vasodilator. Drugs, anesthesia, and contrast medium for angiography were administered through i.v. and intra-arterial (i.a.), catheters. In the hypovolemic model, nine dogs were bled into a reservoir through a catheter passed into the left femoral artery and blood pressure held at 40, 50 or 60 mm Hg for varying time intervals. The peritoneum and intestines were examined after sacrifice at 48 or 72 hr, or at spontaneous death. Normovolemic hypotension was studied in a similar model by injecting isotonic saline into the pericardial sac to reduce the blood pressure to 40 or 50 mm Hg for varying time intervals. Following the shock period, the pericardial fluid was withdrawn and blood pressure normalized. The hearts and intestines were examined either at sacrifice (24 hr) or at spontaneous death. Papaverine infusion (0.5 mg/min) into the superior mesenteric artery was begun at various intervals after the onset of shock and continued throughout the shock period in six dogs with normovolemic shock. Papaverine infusion (0.15 mg/ml, 0.033-0.075 mg/min) into a segmental ileac branch was begun with the onset of normovolemic shock and continued for 30-45 min (2 dogs) or for 2 hr (3 dogs). Arteriograms of the superior mesenteric artery were performed at regular intervals in 9 dogs with hypovolemic hypotension and in 17 dogs with normovolemic hypotension (with and without papaverine). Four of nine dogs died 1.5 to 4.5 hr after the onset of hypovolemic hypotension; ischemic injuries to the small bowel occurred in eight dogs, while the colon was affected in only one dog. On a scale of 0-4, the mean severity of the injuries was 1.67 ± 0.9, and was not affected by the amount and duration of hypotension. Six of 13 dogs died 1-3.5 hr after the onset of normovolemic shock; small bowel and colonic injury were more severe (3.5 ± 0.58) than in hypovolemic hypotension. Normovolemic hypotensive animals given papaverine survived until sacrifice (48 or 72 hr); the mean grade of injury in the 6 dogs receiving papaverine via the superior mesenteric artery was 0.92 ± 0.92, whereas it was 3.31 ± 0.59 in 12 normovolemic hypotensive dogs not given papaverine. Angiography and the post-papaverine vasodilator response did not reflect the nature or extent of injury in either the hypovolemic or normovolemic shock models. However, papaverine administered within 2 hr after the on-

set of normovolemic shock greatly reduced intestinal injury.

1039 THE EFFECT OF DIGITALIS ON REGIONAL ISCHAEMIA OF THE RAT SMALL INTESTINE. (Eng.) Rentzhog, L.; Wikstrom, S. (Univ. Hosp., Uppsala, Sweden). *Upsala J. Med. Sci.* 81(3):179-182; 1976.

Digitoxin was administered to male Sprague-Dawley rats (in which iodine metabolism had been standardized) with induced regional intestinal ischemia to examine its effect on collateral circulation in the small bowel. Exchange circulation was analyzed 30 min after the transmural injection of a test dose of ^{131}INa into the lumen of the isolated intestinal loop. In one experiment, digitoxin (0.1 mg/100 g body weight, i.v.) was administered to 20 rats, and ischemia comprising 11 mesenteric end arcades was established 2 hr after the digitoxin was administered. The exchange circulation was examined 30 min later. The rats retained 50% of the radioactivity in the intestinal loop; whereas untreated ischemic animals and animals without ischemia retained 63 and 25%, resp. The results demonstrate that digitalis disrupts the collateral circulation in adjacent healthy intestine as well as in the small vessels in the intestinal loop. In another experiment, an ischemia comprising seven mesenteric end arcades was induced in 22 rats, followed by administration of digitoxin (0.1 mg/100 g, s.c. daily for 2 wk). The exchange circulation was then analyzed. Four of the treated rats died. After 2 weeks, the weight of rats treated with digitoxin had not changed, whereas untreated ischemic rats had increased their weight by 10%. The treated and untreated groups retained 42 and 37% of the radioactivity in the loop, resp. Disruption of collateral circulation was not observed in this test group, but the increased mortality and the absence of weight increase suggest that prolonged treatment with digitalis may have toxic effects.

1040 CLINDAMYCIN-ASSOCIATED COLITIS IN HAMSTERS: PROTECTION WITH VANCOMYCIN. (Eng.) Bartlett, J. G.; Onderdonk, A. B.; Cisneros, R. L. (Veterans Admin. Hosp., 150 S. Huntington Ave., Boston, MA 02130). *Gastroenterology* 73(4):772-776; 1977.

The possible protective effects of gentamicin, vancomycin, and bacitracin on clindamycin- or lincomycin-induced colitis were investigated in male Syrian hamsters. The animals received lincomycin hydrochloride (2 mg/day, i.m.) or clindamycin phosphate (2 mg/day i.m. or i.p. or 0.1 ml/day, p.o.) for 3 days. Some animals received clindamycin intracecally (5 mg) at laparotomy. Clindamycin or lincomycin was fatal to 65 of 67 recipients during the 2-week observation period. The p.o. administration of gentamicin (2 mg/ml drinking water for 14 days starting 48 hr before initiating lincomycin or clindamycin treatment) prevented mortality in 7 of 11 animals challenged with lincomycin and 5 of 10 animals challenged with clindamycin. The best protective results were achieved with vancomycin and bacitracin. Bacitracin (3 mg/ml drinking water) prevented death in all 10 animals challenged with clindamycin. The simultaneous administration of vancomycin (3 mg/ml) with gentamicin (2 mg/ml) prevented death in all 10 animals challenged with clindamycin and all 9 animals challenged with lincomycin. The administration of vancomycin alone (3 mg/ml, p.o. or 10 mg/day, i.m.) for 14 days prevented death in all 30 animals that received clindamycin. There was no gross or microscopic evidence of intestinal lesions in the animals treated with vancomycin. Clindamycin administration resulted in minimal changes in total aerobic and anaerobic counts, although there was more than a 4-log increase in mean concentrations of coliforms. Vancomycin produced a similar increase in coliform concentrations and a marked decrease in clostridia counts, whereas no change was detected after clindamycin administration. The results demonstrate that vancomycin protects against a lesion in hamsters in which coliform bacteria would be the anticipated pathogens.

1041 ENTERIC FLORA OF NORMAL LABORATORY GUINEA-PIGS. (Eng.) McLean, A. J.; Boquest, A. (Univ. Texas Health Science Center, San Antonio, TX 78284). *Br. J. Exp. Pathol.* 58(3):251-254; 1977.

The bowel flora of guinea pigs was studied using nonselective cultural conditions and conditions highly selective for *Escherichia coli* organisms. No *E. coli* were cultured from adult or juvenile bowel content. Parallel studies showed that culture media supported the growth of small numbers of reference *E. coli* "seeded" into them along with bowel content. It is concluded that normal adult laboratory guinea pigs may be bacteriologically equivalent to the NCS strain of "*E. coli*-free" mice, and it appears, therefore, to represent an experimental model for the study of endotoxin toxicity mechanisms.

1042 ROLE OF COLONIC BACTERIA IN THE PATHOPHYSIOLOGY OF FECAL PERITONITIS. (Eng.) Rink, R. D.; Short, B. L.; Van, N. V.; Fry, D. E. (Univ. Louisville Health Sciences Center, Louisville, KY 40201). *Circ. Shock* 4(3):259-270; 1977.

The role of colonic bacteria in the reactions accompanying fecal peritonitis was investigated in male Sprague-Dawley rats. Septic or nonseptic peritonitis was induced by the administration through an abdominal incision of fresh fecal suspensions or suspensions pretreated with heat or antibiotic. Measurements during 8 hr in rats with septic peritonitis recorded bacteremia, hypoglycemia and progressive hemoconcentration, lactacidemia, and hypocapnea. Mortality was 100% by 24 hr. Nonseptic peritonitis produced significantly lesser degrees of hemoconcentration and hypocapnea. Plasma lactate remained in control ranges, while plasma glucose concentrations increased slightly. Mortality was 5% in 24 hr. The same parameters in control rats were stable over 8 hr. Hepatic oxygen

supply was estimated in each group by multiple platinum wire electrodes. Severe hepatic hypoxia was recorded by 6 hr in rats with septic peritonitis (PO_2=3.0 mm Hg). By contrast, nonseptic peritonitis caused a comparatively mild degree of hypoxia (9.5-10.9 mm Hg) in comparison with that in controls (16.8 mm Hg). The pathophysiologic developments in septic rats appear to be largely the result of hypovolemia induced by colonic bacteria. The administration of a colloid solution (4% Ficol 70 in 0.9% saline) was effective in preventing its development.

1043 EFFECT OF NEONATAL GASTROINTESTINAL COLONIZATION WITH CROSS-REACTING Escherichia coli ON ANTICAPSULAR ANTIBODY PRODUCTION AND BACTEREMIA IN EXPERIMENTAL Haemophilus influenzae TYPE b DISEASE OF RATS. (Eng.) Myerowitz, R. K.; Norden, C. W. (Presbyterian-Univ. Hosp., Pittsburg, PA 15213). *Infect. Immun.* 17(1):83-90; 1977.

Experiments were carried out to determine whether neonatal gastrointestinal colonization by cross-reacting *Escherichia coli* induces protection against experimental *Haemophilus influenzae* type b (HIb) infection. Neonatal gastrointestinal colonization of newborn rats with *E. coli* 075:K100:H5, cross-reactive with the capsular polysaccharide of HIb, was harmless but failed to stimulate detectable (>200 ng/ml) serum anticapsular antibodies. Neonatally colonized rats, when challenged at age 13 weeks by i.p. inoculation of HIb, showed no difference in the frequency, magnitude, or duration of bacteremia or in the postinfection anticapsular antibody response when compared with saline-fed controls. However, neonatally colonized rats challenged at age 4 weeks had a significantly decreased incidence of sustained bacteremia and/or endophthalmitis when compared with controls. This decreased frequency of disease correlated with a significant increase in postinfection serum anticapsular antibodies. Neonatal gastrointestinal colonization with cross-reacting *E. coli* appears to "prime" the young host to respond to infection with HIb with an anticapsular antibody response that protects against sustained HIb bacteremia and its complications.

1044 THYMUS DEPENDENCE AND INDEPENDENCE OF INTESTINAL PATHOLOGY IN A Trichinella spiralis INFECTION: A STUDY IN CONGENITALLY ATHYMIC (NUDE) MICE. (Eng.) Ruitenberg, E. J.; Leenstra, F.; Elgersma, A. (Natl. Inst. Public Health, P.O. Box 1, Bilthoven, Netherlands). *Br. J. Exp. Pathol.* 58(3):311-314; 1977.

The hypothesis that nonspecific histopathological gut changes are thymus-dependent was tested in a *Trichinella spiralis* infection. The study was performed in congenitally athymic (*nu/nu*) mice and their thymus-bearing heterozygous litter mates (+/*nu*). The nonspecific histopathological changes of the small intestine were judged on villus/crypt ratio and mitotic index; for the specific (immunological) reaction the number of pyroninophilic cells in the small intestine was used as criterion. *T. spiralis* induced nonspecific histopathological reactions both in *nu/nu* and +/*nu* mice, whereas the number of pyroninophilic cells was only increased in +/*nu* mice. It is concluded that the immunological reaction was dependent on the immune status of the host, whereas the nonspecific histopathological changes were not (i.e., they were thymus-independent).

1045 EARLY LESIONS OF PANCREATIC DUCTAL CARCINOMA IN THE HAMSTER MODEL. (Eng.) Pour, P.; Althoff, J.; Takahashi, M. (Eppley Inst. for Res. in Cancer, 42nd and Dewey Ave., Omaha, NB 68105). *Am. J. Pathol.* 88(2):291-303; 1977.

The site of pancreatic ductal carcinoma origin was investigated by analyzing the sequence of events in pancreatic carcinogenesis in a hamster model. Syrian golden hamsters were treated s.c. weekly with *N*-nitrosobis(2-oxopropyl)amine (10 mg/kg) for life (group 1) or 6 weeks and were sacrificed at biweekly intervals from 2 weeks (group 1) and 8 weeks (group 2) after initiation of the experiment. The pancreas was examined in step sections, and the sequential alterations noted for each interval were recorded. Lesions were found in intrapancreatic and extrapancreatic ducts. Equivalent alterations consisting of hyperplasia, metaplasia, atypia, and lesions characteristic of carcinoma *in situ* developed ubiquitously and simultaneously in pancreatic ducts of different sizes and in ductules, but not in acinar cells. Among the most significant findings were intrainsular ductular formations, their proliferation, and sequential malignant alteration comparable to the involved pre-existing ductules. Differences between the two experimental groups were of a quantitative rather than qualitative nature. The results indicate a common origin of all induced tumors from a pluripotent cell populating the pancreatic ductal system.

1046 HYPEROSMOLALITY AND PANCREATIC BLOOD FLOW. (Eng.) Jarhult, J.; Thulin, A. (Dept. Physiology, Univ. Lund, Solvegatan 19, S-22362 Lund, Sweden). *Pfluegers Arch.* 370(2):127-130; 1977.

The possible role of regional hyperosmolality in functional pancreatic hyperemia was studied in the cat pancreas. Close intra-arterial infusions of hypertonic xylose, glucose, and sucrose solution (30-50%) caused a marked vasodilatation but no secretion. The magnitude of the vasodilator response correlated with the degree of induced glandular hyperosmolality but not with the substance per se. The initial decrease of vascular resistance (peak response) averaged about 35% of the control value, whereas the steady-state response was 60-70% of the control value. Stimulation of the vagus nerve as well as infusion of secretin and/or cholecystokinin evoked a pronounced secretion and a small to moderate increase in pancreatic blood flow (peak increase 130-225% of control), but did not significantly change the plasma osmolality in the venous effluent from the

gland. Bradykinin, papaverine, and isoproterenol caused large increments of pancreatic blood flow; none of these drugs evoked secretion, nor did they change the venous osmolality. It is concluded that blood-borne hyperosmolality is a strong stimulus for pancreatic vasodilatation, but the functional hyperemia of this gland is due to factors other than regional tissue hyperosmolality.

1047 SELF-REGULATION OF REGIONAL CIRCULATION IN THE PANCREAS. EFFECT OF PERIPHERAL VENOUS INTRAMURAL STIMULATION. 4. INFLUENCE OF ATROPINE. (Ita.) Musca, A.; Marigliano, V.; Cordova, C.; Cammarella, I. (VI Cattedra di Semeiotica Medica, Universita di Roma, Rome, Italy). *Boll. Soc. Ital. Biol. Sper.* 52(16):1215-1218; 1976.

The influence of atropine on pressor valve response was studied in the pancreas of the dog. Percentage variation in pancreatic circulation was measured at 15-min intervals beginning at 30 min and continuing for 3 hr after i.v. administration of 30 µg/kg atropine sulfate. Peak variations from baseline values occurred between 30 min and 2 hr after treatment. Atropine sulfate is capable of interrupting pressor response at the point of expression of the vascular region of the pancreas but not in the intramural peripheral veins.

1048 SELF-REGULATION OF REGIONAL CIRCULATION IN THE PANCREAS. EFFECT OF PERIPHERAL VENOUS INTRAMURAL STIMULATION. 5. INFLUENCE OF SODIUM PENTOBARBITAL. (Ita.) Marigliano, V.; Musca, A.; Cordova, C.; Cammarella, I. (VI Cattedra di Semeiotica Medica, Universita di Roma, Rome, Italy). *Boll. Soc. Ital. Biol. Sper.* 52(16):1219-1222; 1976.

The influence of sodium pentobarbital on pressor valve response was studied in the pancreas of the dog. Percentage variation in pancreatic circulation was measured at 15-min intervals beginning at 30 min and continuing for 3 hr after i.v. administration of 30 µg/kg sodium pentobarbital. Sodium pentobarbital interrupts pressor response at the point of expression of the vascular regions of the pancreas but not in the peripheral veins.

1049 EXPERIMENTAL STUDY OF THE SELF-REGULATION OF REGIONAL CIRCULATION IN THE PANCREAS. EFFECT OF PERIPHERAL VENOUS INTRAMURAL STIMULATION. 6. INFLUENCE OF HISTAMINE. (Ita.) Musca, A.; Marigliano, V.; Cordova, C.; Cammarella, I. (VI Cattedra di Semeiotica Medica, Universita di Roma, Rome, Italy). *Boll. Soc. Ital. Biol. Sper.* 52(16): 1223-1228; 1976.

The influence of histamine on pressor valve response was studied in the pancreas of the dog. Percentage variation in pancreatic circulation was measured at 15-min intervals beginning at 30 min and continuing for 3 hr after i.v. administration of 30 µg/kg histamine. Histamine does not inhibit pressor response in the peripheral veins but rather intensifies it. Pancreatic circulation increases with time in a prolongation of the phenomenon after administration of the drug.

1050 PANETH CELLS IN EXPERIMENTAL AMYLOIDOSIS. (Eng.) Ahonen, A.; Penttila, O.; Klinge, E. (Dept. Pharmacology, Univ. Helsinki, Siltavuorenpenger 10 A, SF-00170 Helsinki 17, Finland). *Scand. J. Gastroenterol.* 12(2):129-133; 1977.

The effect of experimental amyloidosis on the morphology of mouse Paneth cells was investigated, using light and fluorescence microscopy. Amyloidosis was induced by daily s.c. casein injections (0.2 ml) for 4 weeks. The number of secretory granules of the Paneth cells increased in the jejunum ($p<0.001$), but remained normal in the duodenum. Small morphological alterations took place in the Paneth cells during the induction of amyloidosis, and occasionally the Paneth cells were totally lost. These changes suggest that the function of the Paneth cells of the jejunum may be partly inhibited during experimental amyloidosis.

1051 PURIFICATION OF HOG GASTRIC INTRINSIC FACTOR BY A SIMPLE TWO-STEP PROCEDURE BASED ON AFFINITY CHROMATOGRAPHY AND A SELECTIVE GUANIDINE HYDROCHLORIDE GRADIENT. (Eng.) Francis, G. L.; Smith, G. W.; Toskes, P. P.; Sanders, E. G. (Veterans Admin. Hosp., Gainesville, FL 32602). *Gastroenterology* 72(6):1304-1307; 1977.

A method for the separation of intrinsic factor (IF) from NIF (a vitamin B_{12}-binding glycoprotein not active in promoting vitamin B_{12} absorption) is described. Monocarboxylic acid derivatives of vitamin B_{12} were covalently coupled to 1,6-hexanediamine-substituted Sepharose by using a water-soluble carbodiimide resulting in 1.32 µmol of B_{12} coupled per ml of Sepharose. After a source of crude hog IF was passed over the column, a selective linear gradient of guanidine HCl (0-4.0 M) was used to remove IF and 4.0-7.5 M to elute NIF. Anti-IF antibodies blocked 99% of the B_{12} binding by the isolated IF and only 1% of the B_{12} binding by NIF. Passage over a hydroxyapatite column resulted in IF 99% pure with a specific activity of 29.8 µg of B_{12} binding per mg of protein. IF so isolated exhibited one homogeneous band on polyacrylamide gel electrophoresis and corrected B_{12} malabsorption in a patient with pernicious anemia.

1052 ANTHELMINTIC DIHYDROQUINOXALINO[2,3-b]-QUINOXALINES. (Eng.) Fisher, M. H.; Lusi, A.; Egerton, J. R. (Merck and Co., Inc., Rahway, NJ 07065). *J. Pharm. Sci.* 66(9):1349-1352; 1977.

1053 MORPHOLOGIC DEVELOPMENT OF THE HUMAN FETAL SMALL INTESTINE. (Eng.) Moxey, P. C. *In: Gastrointestinal Development and Neonatal Nutrition. Report of the Seventy-second Ross Conference on Pediatric Research held by the Harvard Medical School Department of Pediatrics at*

Proust's Neck, Maine, June 13-16, 1976. (Columbus: Ross Laboratories):19-23; 1977.

1054 CHANGES IN THE DIGESTIVE AND OTHER ORGANS OF PIGS IN THE PERINATAL PERIOD. (Eng.) Widdowson, E. M. *In: Gastrointestinal Development and Neonatal Nutrition. Report of the Seventy-second Ross Conference on Pediatric Research held by the Harvard Medical School Department of Pediatrics at Proust's Neck, Maine, June 13-16, 1976.* (Columbus: Ross Laboratories):35-40; 1977.

1055 EPITHELIAL-MESENCHYMAL INTERACTIONS IN GASTROINTESTINAL ORGAN DEVELOPMENT. (Eng.) Bernfield, M. *In: Gastrointestinal Development and Neonatal Nutrition. Report of the Seventy-second Ross Conference on Pediatric Research held by the Harvard Medical School Department of Pediatrics at Proust's Neck, Maine, June 13-16, 1976.* (Columbus: Ross Laboratories):40-44; 1977.

1056 FUNCTIONAL ADAPTATION OF EXOCRINE GLANDS. (Eng.) Grand, R. J. *In: Gastrointestinal Development and Neonatal Nutrition. Report of the Seventy-second Ross Conference on Pediatrics Research held by the Harvard Medical School Department of Pediatrics at Proust's Neck, Maine, June 13-16, 1976.* (Columbus: Ross Laboratories):59-62; 1977.

1057 NON-STEROID ANTI-INFLAMMATORY DRUGS: COMBINED ASSAY FOR ANTI-EDEMIC POTENCY AND GASTRIC ULCEROGENESIS IN THE SAME ANIMAL. (Eng.) Rainsford, K. D.; Whitehouse, M. W. (Dept. Biochemistry, Univ. Tasmania, Hobart 7001, Australia). *Life Sci.* 21(3):371-377; 1977.

1058 EFFECTS OF ASPIRIN-LIKE DRUGS ON CANINE GASTRIC MUCOSAL BLOOD FLOW AND ACID SECRETION. (Eng.) Bennett, A.; Curwain, B. P. (King's Coll. Hosp. Medical Sch., London SE5 8RX, England). *Br. J. Pharmacol.* 60(4):499-504; 1977.

1059 THE ROLE OF TOPICALLY APPLIED DEXAMETHASONE IN PREVENTING PERITONEAL ADHESIONS: EXPERIMENTAL AND CLINICAL STUDIES. (Eng.) Shikata, J. I.; Yamaoka, I. (Teikyo Univ. Sch. Medicine, 2-11-1 Kaga, Itabashi, ku, Tokyo 173, Japan). *World J. Surg.* 1(3):389-395; 1977.

1060 BILE INDUCED CHRONIC GASTRIC ULCER IN SWINE. (Eng.) Rokkjaer, M.; Sogaard, H.; Kruse, A.; Amdrup, E. (Kommunehospitalet, DK-8000 Aarhus C, Denmark). *World J. Surg.* 1(3):371-379; 1977.

1061 SECRETORY IgA ANTIBODIES AGAINST COW'S MILK PROTEINS IN HUMAN MILK AND THEIR POSSIBLE EFFECT IN MIXED FEEDING. (Eng.) Hanson, L. A.; Ahlstedt, S.; Carlsson, B.; Fallstrom, S. P. (Inst. Medical Microbiology, Guldhedsgatan 10, S-413 46 Goteborg, Sweden). *Int. Arch. Allergy Appl. Immunol.* 54(5):457-462; 1977.

1062 MYOELECTRICAL ACTIVITY OF THE CANINE STOMACH AND DUODENUM AFTER GASTRIC RESECTION. (Eng.) O'Leary, J. F.; Hennessy, T. P. J. (Dept. Surgery, Univ. Coll., Cork, Ireland). *J. Surg. Res.* 23(3):161-165; 1977.

1063 DEPENDENCE OF HORMONAL EFFECTS FROM NUTRIENTS SUPPLY. (Eng.) Nesterin, M. F. (USSR Acad. Medical Sciences, Moscow, USSR). *Nutr. Metab.* 21(Suppl. 1):249-253; 1977.

1064 AGE-SPECIFIC DIFFERENTIATION DURING THE DEVELOPMENT OF THE RAT SUBMANDIBULAR GLAND. (Eng.) Denny, P. C.; Cope, P. (Ethel Percy Gerontology Center, Los Angeles, CA). *Differentiation* 8(2):105-111; 1977.

1065 COMPARATIVE DEVELOPMENTAL ASPECTS OF SELECTED ORGAN SYSTEMS. II. GASTROINTESTINAL AND UROGENITAL SYSTEMS. (Eng.) Hoar, R. M. (Hoffmann-La Roche Inc., Nutley, NJ 07110). *Environ. Health Perspect.* 1:61-66; 1976.

1066 NUTRITIONAL EFFECTS OF SALMONELLOSIS IN MICE. (Eng.) Moore, R. N.; Johnson, B. A.; Berry, L. J. (Dept. Microbiology, Univ. Texas, Austin, TX 78712). *Am. J. Clin. Nutr.* 30(8):1289-1293; 1977.

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1068 INFLUENCES OF ULCEROGENIC STIMULATIONS ON THE STOMACH WITH THE CLAMPING ULCERS IN RATS. (Eng.) Miyazawa, M. (Tokyo Medical Coll., 7-1 Nishi-Shinjuku, 6-chome, Shiujuku-ku, Tokyo 160, Japan). *Gastroenterol. Jpn.* 12(2):8-19; 1977.

1069 ACTIVITIES OF INTESTINAL ENZYMES IN EXPERIMENTAL CHRONIC RENAL INSUFFICIENCY. (Eng.) Grimmel, K.; Bongartz, S.; Kaspar, H. (Medizinische Universitatsklinik Wurzburg, D-8700 Wurzburg, W. Germany). *Nephron* 19(2):81-87; 1977.

1070 CARRAGEENAN IN FORMULA AND INFANT BABOON DEVELOPMENT. (Eng.) McGill, H. C., Jr.; McMahan, C. A.; Wigodsky, H. S.; Sprinz, H. (Univ. Texas Health Sciences Center, 7703 Floyd Curl Drive, San Antonio, TX 78284). *Gastroenterology* 73(3):512-517; 1977.

1071 THE EFFECTS OF VARIOUS FORMS OF GASTRO-INTESTINAL CANNULATION ON DIGESTIVE MEASUREMENTS IN SHEEP. (Eng.) MacRae, J. C.; Wilson, S. (Hill Farming Res. Organisation, Bush Estate, Penicuik, Midlothian EH26 OPY, England). *Br. J. Nutr.* 38(1):65-71; 1977.

1072 INTESTINAL LESIONS INDUCED IN GNOTOBIOTIC CALVES BY THE VIRUS OF HUMAN INFANTILE GASTROENTERITIS. (Eng.) Mebus, C. A.; Wyatt, R. G.; Kapikian, A. Z. (Dept. Veterinary Science, Univ. Nebraska, Lincoln, NB 68583). *Vet. Pathol.* 14(3): 273-282; 1977.

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1075 PRODUCTION OF INTESTINAL AND OTHER TUMORS BY 1,2 DIMETHYLHYDRAZINE DIHYDROCHLORIDE IN MICE. I. A LIGHT AND TRANSMISSION ELECTRON MICROSCOPIC STUDY OF COLONIC NEOPLASMS [Abstract]. (Eng.) Toth, B.; Malick, L.; Shimizu, H. (Univ. Nebraska Medical Center, Omaha, NB 68105). *Am. J. Pathol.* 84(1):69-86; 1976.

1076 CARCINOGENS OCCURRING NATURALLY IN FOODS. (Eng.) Miller, J. A.; Miller, E. C. (Univ. Wisconsin Medical Center, Madison, WI 53706). *Fed. Proc.* 35(6):1316-1321; 1976.

1077 PORTAL PRESSURE REDUCTION INDUCED BY BALLOON OBSTRUCTION OF THE SUPERIOR MESENTERIC ARTERY (SMA) [Abstract]. (Eng.) Groszmann, R. J.; Blei, A. T.; Kniaz, J. L.; Storer, E. H.; Conn, H. O. (Veterans Admin. Hosp., West Haven, CT). *Gastroenterology* 73(5):1222; 1977.

1078 THE ROLE OF LIPIDS IN THE DIETARY REGULATION OF PLASMA BILIRUBIN CONCENTRATION [Br] IN GUNN RATS [Abstract]. (Eng.) Billing, B. H.; Hole, D. R.; Tickner, T. R.; Gollan, J. L. (Royal Free Hosp., London, England). *Gastroenterology* 73(5):1213; 1977.

1079 EFFECT OF THE HISTAMINE H2 ANTAGONIST CIMETIDINE ON GASTRIC MUCOSAL BLOOD FLOW [Abstract]. (Eng.) Knight, S. E.; McIsaac, R. L.; Fielding, L. P. (St. Mary's Hosp. Medical Sch., London, England). *Gut* 18(11):A948; 1977.

1080 MUCOSAL ADHERENCE OF HUMAN ENTEROPATHOGENIC E. coli (EPEC) IS MEDIATED BY A TRANSMISSIBLE PLASMID [Abstract]. (Eng.) McNeish, A. S.; Williams, P. H.; Evans, N.; Turner, P.; George, R. H. (Univ. Leicester, Leicester, England). *Gut* 18(11):A943; 1977.

1081 INFLUENCE OF METRONIDAZOLE AND NEOMYCIN ON COLONIC MICROFLORA IN HEALTHY VOLUNTEERS [Abstract]. (Eng.) Arabi, Y.; Dimock, F.; Burden, D. W.; Alexander-Williams, J.; Keighley, M. R. B. (General Hosp., Birmingham, England). *Gut* 18(11):A969-A970; 1977.

See also, 0785, 0861, 0967, 1013, 1621.

DIAGNOSTIC PROCEDURES

1082 EMERGENCY DIAGNOSIS OF UPPER GASTROINTESTINAL BLEEDING BY FIBEROPTIC ENDOSCOPY. (Eng.) Villar, H. V.; Fender, H. R.; Watson, L. C.; Thompson, J. C. (Univ. Texas Medical Branch, Galveston, TX 77550). *Ann. Surg.* 185(3):367-374; 1977.

Experience with 192 patients in whom emergency esophagogastroduodenoscopy was used in the diagnosis and treatment of massive upper gastrointestinal bleeding is presented. Accurate endoscopic diagnosis was made in 184; 58 patients underwent emergency operations to control bleeding with an overall operative mortality of 26%. Excluding 16 patients who underwent emergency portacaval shunting, the operative mortality was 7%. In six patients, the bleeding was controlled by endoscopic electrocoagulation. There were no complications. Emergency endoscopy should be done routinely as the primary diagnostic approach in the diagnosis of upper gastrointestinal bleeding.

1083 ELECTRICAL ASSESSMENT OF FUNCTIONAL LACTASE ACTIVITY IN CONSCIOUS MAN. (Eng.) Read, N. W.; Davies, R. J.; Holdsworth, C. D.; Levin, R. J. (Dept. Medicine, Univ. Sheffield, Sheffield S10 2TN, England). *Gut* 18(8):640-643; 1977.

The hypothesis that the magnitude of the lactose potential may reflect the functional lactase activity in the small intestine was tested. Using an electrical technique for measuring transjejunal potential differences (PDs) in conscious man, the electrogenic absorption of the hexoses liberated by hydrolysis of lactose was estimated. Lactose was infused into the jejunum of 1 normal control and 21 patients with diarrhea. The results were compared with jejunal lactase levels determined from biopsy specimens taken from the recording site immediately after infusion. The PD evoked by 100 mM lactose was significantly lower in patients with lactase levels below 4 U (lactase deficient) compared with subjects with normal lactase levels. There was also a significant correlation ($r=0.87$, $p<0.005$) between the magnitude of the lactose potential (expressed as the ratio of the maximum glucose transfer potential) and the mucosal lactase level in the hypolactasic subjects, but not in patients with normal lactase levels. Thus, in the subjects with lactase deficiency, the electrogenic transfer of hexose is clearly limited by the rate of lactose hydrolysis. Unlike other assessments of functional lactase activity, the electrical test provides a specific index of jejunal function and, moreover, can be adapted to investigate the possible disorders of small intestinal motility and secretion associated with hypolactasia.

1084 $^{14}CO_2$ EXHALATION TEST. A NEW DIAGNOSTIC TOOL IN GASTROENTEROLOGY. (Ger.) Adlung, J.; Kelch, L.; Pancho, W.; Grazikowske, H.; Uthgennant, H. (I. Medizinische Klinik, Medizinische Hochschule Lubeck, Kronsforder Allee 71/73, D-2400 Lubeck, W. Germany). *Z. Gastroenterol.* 14(5):565-573; 1976.

The diagnostic value of three $^{14}CO_2$ exhalation tests (^{14}C-tripalmitate fat resorption test, ^{14}C-lactose resorption test, and ^{14}C-glycocholic acid test) was studied in normal subjects and in patients with gastrointestinal diseases. The tripalmitate resorption test was performed in 20 normal subjects, in 18 patients with chronic recurrent pancreatitis without manifest pancreatic insufficiency, and in 8 patients with advanced malassimilation syndrome. The percentage of $^{14}CO_2$ exhaled in 8 hr by the normal subjects averaged 9.1%. The $^{14}CO_2$ exhalation was significantly reduced only in the patients with advanced malassimilation syndrome. The percentage of $^{14}CO_2$ exhaled in 4 hr in the lactose resorption test averaged 11.9 ± 0.6% in 27 healthy controls, showing no significant difference in 16 patients with and without lactase deficiency who had undergone Billroth-II resection, nor in 6 patients with malabsorption and lactase deficiency. The glycocholic acid test was performed in 22 normal subjects, 8 patients with suspected bacterial infection of the small intestine, and in 11 patients with resected ileum or Crohn's disease. Significantly decreased $^{14}CO_2$ exhalation was found in 16/19 patients, but false-negative results were obtained for 3 patients with resected ileum. Differentiation between bile salt malabsorption and bacterial infection was, however, impossible. The findings indicate the small value of the $^{14}CO_2$ exhalation tests in gastroenterological disorders. Simplicity and convenience alone are not enough to recommend these tests.

1085 RADIOLOGICAL DIAGNOSIS OF TUMORS OF THE SMALL INTESTINE. (Ger.) Scherer, K.; Bucheler, E.; Erbe, W. (Radiologische Klinik, Universitatskrankenhaus Hamburg-Eppendorf, Martinistrasse 52, 2000 Hamburg 20, W. Germany). *Roentgenblaetter* 30(8):379-387; 1977.

The possibilities for radiologic diagnosis of tumors of the small intestine are reviewed. Barium x-ray; pharmacoradiological methods with cerulein; enteric-coated capsules of tartaric acid, sodium hydrogen carbonate, and dimethylpolysiloxane; Pro-Banthine or Buscopan; angiographic methods; and especially Sellink's probing method are suitable for the diagnosis of small intestinal tumors. The fractionated gastrointestinal passage method is problematic due to the possible superposition of intestinal loops in the small pelvis, and to the high time requirement for the examination. Angiography is especially suitable for the diagnosis of vascularized tumors with extramural spread, such as leiomyoma, neurinoma, hemangioma, and even carcinoid and duodenal carcinoma, despite their low degree of vascularization. Such poorly vascularized tumors as adenomas, lipomas, fibromas, neurofibromas, adenocarcinomas, metastases, and lymphoreticular sarcomas require intestinal passage tests for diagnosis.

1086 CARCINOEMBRYONIC ANTIGEN IN COLONIC LAVAGE. (Eng.) Winawer, S. J.; Fleisher, M.; Green, S.; Bhargava, D.; Leidner, S. D.; Boyle, C.; *et al.* (Memorial Hosp., 1275 York Ave., New York,

NY 10021). *Gastroenterology* 73(4, Part 1):719-722; 1977.

Colonoscopic lavage was performed for assay of carcinoembryonic antigen (CEA) concentration in 35 patients with small and large colonic adenomas, in 11 patients with adenocarcinoma of the colon, and in 12 normal controls. CEA analyses were carried out in perchloric acid extracts of both colonic and plasma specimens by Sephadex G-200 gel column chromatography and electrophoresis. CEA concentration was considerably higher in colonoscopic lavage as compared to plasma. CEA concentration in normal controls (266 ng/mg protein) and in patients with colonic adenomas <1 cm in size (372 ng/mg) were not significantly different. CEA concentrations in patients with colonic adenomas >1 cm in size (1,151 ng/mg) and in patients with adenocarcinoma of the colon (1,509 ng/mg) were significantly higher than those observed in patients with small adenomas and in the normal controls. In all except two patients, plasma CEA concentrations were normal. Assay of CEA concentrations in colonic lavage may have potential application in the further assessment of the mucosa at risk for colon adenomas and for colon cancer. It does not appear to be accurate enough for diagnosis of colon cancer in individual patients.

1087 ENDOSCOPY AND POLYPECTOMY FOR THE EARLY DETECTION OF CARCINOMA OF THE RECTUM AND THE COLON (METHODS, SIGNIFICANCE, AND LIMITATIONS). (Ger.) Otto, P.; Helmstaedt, D. (Medizinische Abteilung, Kreiskrankenhaus Grossburgwedel, 3006 Burgwedel 1, W. Germany). *Roentgenblaetter* 30(8): 400-407; 1977.

The state of the art of endoscopy in the early diagnosis and treatment of colorectal polyps and carcinomas is described. As routine rectoscopy is not justified in screening, it should be restricted to patients with occult blood in the feces and suspect anamnesis (perianal bleeding, irregular bowel movements, and abdominal spasms). If carcinoma is found rectoscopically, double-contrast x-ray examination should be performed in view of the possibility of double carcinoma. Coloscopy is necessary, if the source of the bleeding is not detected by rectoscopy or x-ray examinations. High coloscopy should be reserved for use in clinics, while sigmoidoscopy can be performed routinely. All colorectal polyps should be completely removed for correct histological diagnosis. Endoscopic polypectomy is an effective curative therapy, and surgery is necessary only in the case of polyps invading the muscular layer. In a series of 18,814 diagnostic and 3,490 therapeutic coloscopies, the mortality rate was 0.026% versus 1-1.5% after transabdominal colostomy with polypectomy.

1088 ARTERIOGRAPHY IN MALIGNANT NEOPLASMS OF THE BOWEL. (Eng.) Campbell, D. R.; Mason, W. F.; Fraser, D. B.; Standen, J. R. (Victoria General Hosp., Halifax, Nova Scotia B3H 2Y9, Canada). *J. Can. Assoc. Radiol.* 28(3):204-207; 1977.

Experience with arteriography in the investigation of gastrointestinal bleeding and in the diagnosis of malignant bowel neoplasms is presented. Eighty patients with gastrointestinal bleeding were investigated by angiography over a 4-yr period. Nine had malignant neoplasms of the bowel, and the case reports of four of these patients are presented. Inconspicuous neovascularity was the most constant finding, best seen in leiomyosarcoma of the small bowel and adenocarcinoma of the colon. Neovascularity was not seen in a patient with reticulum cell sarcoma of colon, detected by extravasation. In two of the patients whose case reports are presented, the malignancies were missed by standard barium studies; these lesions involved the jejunum and ascending colon, resp. When other studies including endoscopy and barium examinations have failed to explain acute or chronic bleeding, an appreciable number of intestinal neoplasms may be demonstrated by arteriography.

1089 "HOT SPOT" ON HEPATIC SCINTIGRAPHY AND RADIONUCLIDE VENACAVOGRAPHY. (Eng.) Gooneratne, N. S.; Buse, M. G.; Quinn, J. L., III; Selby, J. B. (Medical Univ. South Carolina Hosp., 80 Barre St., Charleston, SC 29401). *Am. J. Roentgenol.* 129(3):447-450; 1977.

Seven patients showing a localized area of increased uptake (hot spot) on ^{99m}Tc sulfur colloid liver scans are described. Dynamic studies were also carried out in four of the patients. The hot spot was associated with superior vena caval obstruction in five patients, with inferior vena caval obstruction in one, and with a hepatoma in one. Clinical evidence of vena caval obstruction was present in only three cases. Although a hot spot on static liver images usually indicates superior vena caval obstruction, the addition of radionuclide venacavography is recommended to confirm the diagnosis. A hot spot in the liver area in a radionuclide venogram of the lower limbs suggests inferior vena caval obstruction with collaterals via the liver.

1090 RADIOLOGIC SPECTRUM OF MELANOMA METASTATIC TO THE GASTROINTESTINAL TRACT. (Eng.) Goldstein, H. M.; Beydoun, M. T.; Dodd, G. D. (M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030). *Am. J. Roentgenol.* 129(4):605-612; 1977.

Radiologic experience with 67 cases of metastatic melanoma involving the gastrointestinal tract is presented. The areas of involvement included the esophagus (5 cases), stomach (16 cases), duodenum (13 cases), small intestine (32 cases), colon (3 cases), liver (10 cases), gallbladder (3 cases), biliary tree and pancreas (4 cases), and mesentery and omentum (12 cases). Three patients with gastroduodenal lesions had a solitary nodule in the stomach that was detected only by double contrast examination. The intramural nodule was the most commonly encountered melanoma lesion in the stomach and duodenum. The nodules varied considerably in size. Approximately an equal number were present with and without a central ulceration, and both

types of nodules were frequently encountered in the same patient. In two instances, gastric tumors also had a large extragastric component. The largest diversity of radiologic presentations of melanoma metastases occurred in the small intestine. A frequent manifestation was also one or more mural nodules. The "target" appearance occurred much less frequently in the small intestine than in the stomach and duodenum. In one case, innumerable melanoma nodules were present that resembled a polyposis syndrome. Seven patients demonstrated a large excavating mass in the mid or distal small bowel. In four cases, the entire mass was ulcerated. Angiography revealed a variety of vascular patterns of liver metastases; these ranged from hypervascular to totally hypovascular. Involvement of biliary ducts and pancreatic area was demonstrated primarily by percutaneous transhepatic cholangiography when jaundice was present. Biliary obstruction occurred in the porta hepatis region as well as at the distal common bile duct by peripancreatic nodes. Mesenteric masses were often purely extrinsic, causing impression upon and displacement of bowel loops. Because of extended survival due to improved chemotherapy and immunotherapy, the importance of careful radiologic examination of the melanoma patient is stressed.

1091 GRAY SCALE ULTRASONOGRAPHY OF CALCIFIED LIVER METASTASES. (Eng.) Katragadda, C. S.; Goldstein, H. M.; Green, B. (M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030). *Am. J. Roentgenol.* 129(4):591-593; 1977.

Three cases of calcified liver metastases demonstrating acoustical shadowing on gray scale ultrasonography are reported. The patients had metastases from adenocarcinomas of the rectosigmoid, cecum, and splenic flexure, resp. Plain abdominal radiographs demonstrated the calcifications, but they were not readily apparent in most cases. It is possible that gray scale ultrasonography will disclose calcified metastases in a patient with a false-negative radiograph.

1092 ULTRASONIC SCANNING FOR FOCAL LIVER LESIONS. (Eng.) Rasmussen, S. N.; Bagger, J.; Wagner, F.; Pedersen, J. F.: Kristensen, J. K. (Blegdamshospitalet, Blegdamsvej 3, DK-2200 Copenhagen N, Denmark). *Dan. Med. Bull.* 24(4): 151-153; 1977.

The diagnostic accuracy of ultrasonic bistable scanning for focal liver lesions was evaluated in 157 patients. Using the information from the patients' charts up to the date of the ultrasonic examination, these 157 cases were divided into two categories: (A) patients strongly suspected of having focal liver lesions and (B) patients only slightly suspected of having focal liver lesions. In group A the predictive values of positive (lesion present) and negative (no lesion present) ultrasonic findings were 81% (62-94%) and 33% (10-65%), resp., with 95% confidence limits in brackets. In group B the corresponding values were 74% (51-90%) and 64% (52-75%), resp. It is concluded that in group A no diagnostic profit was obtained by the ultrasonic examination, while some diagnostic profit was obtained in group B. The diagnostic accuracy increased with an increasing number of focal liver lesions and with increasing size of the lesions.

1093 A COMPARISON OF GRAY SCALE ULTRASOUND AND RADIONUCLIDE IMAGING FOR THE DETECTION OF FOCAL HEPATIC LESIONS: OPEN SHUTTER TECHNIQUE. (Eng.) Zatz, L. M.; Gouldin, J. A.; Hanley, G. A. (Veterans Admin. Hosp [114], Palo Alto, CA 94304). *J. Clin. Ultrasound* 5(3):178-184; 1977.

The results of a new, previously described, gray scale ultrasound technique were compared with those of radionuclide imaging to determine if the former technique could replace the latter in the detection of focal hepatic lesions. The conventional B scan ultrasound unit was modified by the addition of a specially built logarithmic amplifier and a high resolution nonstorage oscilloscope. One hundred and five consecutive patients submitted for radionuclide imaging of the liver were examined with ultrasound using a standardized scanning system. The ultrasound technique produced scans that were technically poor or inadequate in 37% of the patients studied. In the studies in which there has been confirmation, the ultrasound diagnosis was accurate in 73% and the radionuclide diagnosis was correct in 83%. The combined accuracy of the two studies was 93%. In 11 patients, ultrasound more clearly defined the lesions that were demonstrated by both techniques or demonstrated the nature of suspicious or equivocal areas on the radionuclide images. In 48 patients, the radionuclide scan imaged the liver more completely or demonstrated lesions more clearly than did the ultrasound examination. This specific approach to ultrasound gray scale imaging appears to be a valuable supplemental tool to radionuclide imaging, but at its present stage of development, cannot replace it as a routine screening technique for hepatic disease.

1094 THE INTERPRETATION OF THE RADIONUCLIDE SUBTRACTION SCAN IN PANCREATIC CARCINOMA. (Eng.) Andrews, J. T.; Kidd, G.; Steven, L. W.; McKay, W. J.; Lichtenstein, M. (Royal Melbourne Hosp., Melbourne, Australia). *Australas. Radiol.* 21(1):53-59; 1977.

An attempt was made to indicate the different types of radionuclide subtraction scan patterns that can occur in pancreatic carcinoma. Forty-four consecutive patients with established clinical or proven histological diagnosis of carcinoma of the pancreas were followed from a total series of 800 who presented for radionuclide subtraction scanning. Over one-third of the patients studied presented with ^{75}Se-selenomethionine uptake in the tumor region. The tumor was diagnosed because of the presence of a filling defect in an otherwise normal pancreatic scan in only four cases. Despite the differing scan presentations of pancreatic carcinoma, it was generally not possible to differentiate a benign from a malignant lesion on the basis of the scan results alone.

1095 OPERATIVE CHOLANGIOGRAPHY. NEW CHOLANGIOGRAM CATHETER CLAMP AND IMPROVED TECHNIQUE. (Eng.) Borge, J. (Queen of the Valley Hosp., 980 Traneas St., Napa, CA 94558). *Arch. Surg.* 112(3): 340-342; 1977.

Experience with 225 cases of routine operative cholangiography, utilizing a new squeeze-locking, split-loop clamp to secure the catheter inside the cystic duct plus an x-ray film technique using two different dye concentrations is presented. No surgical or procedural difficulty was experienced in any of the 225 cases. Additional operating time for the procedure averaged under 5 min, with only one inconclusive x-ray film study. These results suggest that the new catheter clamp and the two-film technique provide a mechanical simplification of the procedure and that many of the causes of inconclusive x-ray film study have been eliminated.

1096 AN IMPROVED METHOD OF EXAMINING THE STOMACH BY THIN-COATING DOUBLE-CONTRAST TECHNIQUE. (Ger.) Tenner, R.; Kurosawa, A. (Tinsdaler Weg 21, 2000 Wedel/Holst., W. Germany). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 125(6): 490-496; 1976.

1097 PHARMACOKINETIC INVESTIGATIONS OF A NEW GALLBLADDER CONTRAST MEDIUM--IODOXAMINIC ACID. (Ger.) Mahner, B.; Kruger, R.; Siehr, U.; Wilhelmi, U. (Stadt. Krankenhaus Spandau, 1000 Berlin 20-Spandau, W. Germany). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 125(6): 556-559; 1976.

1098 A RAPID METHOD OF ROENTGENOLOGIC EXAMINATION OF THE SMALL INTESTINE WITH BUSCOPAN®. (Jpn.) Watanabe, T.; Ohata, T.; Izuno, I. (Sch. Medicine, Shinshu Univ., Japan). *Nippon Acta Radiol.* 36(4):305-308; 1976.

1099 THE USE OF UROLOGICAL CATHETERS FOR THE SURGICAL EXPLORATION OF THE BILIARY TRACT. (Spa.) Bravo, J. L.; Vazquez, E.; Alberu, J. (Instituto Nacional de la Nutricion, Mexico 22, D.F.). *Rev. Invest. Clin.* 29(1):9-11; 1977.

1100 ELIMINATION OF INDOCYANINE GREEN (ICG) IN LIVER DISEASES AFTER INCREASED DYE LOADING AND PROLONGED TIME OF MEASUREMENTS. (Pol.) Juszczyk, J.; Grala, B.; Adamek, J.; Kryska, A.; Panczyszak, M. (Acad. Medicine, ul. Wincentego 1, 61-003 Poznan, Poland). *Pol. Arch. Med. Wewn.* 56(2/8):119-132; 1976.

1101 PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY WITH THE CHIBA NEEDLE. (Dut.) Yap, S. H.; Snel, P.; Rosekrans, P. A. M.; Wiggers, R. H.; Van Tongeren, J. H. M. (St. Radboud Ziekenhuis, Nijmegen, Netherlands). *Ned. Tijdschr. Geneeskd.* 121(3):96-100; 1977.

1102 THERMOVISION AS A DIAGNOSTIC METHOD IN GASTROENTEROLOGY. (Rus.) Sukharev, V. F. (I. I. Dzhanelidze Scientific Res. Inst. First Aid, Leningrad, USSR). *Sov. Med.* (12):98-101; 1976.

1103 THE PHYSICS OF THE FIBRE-OPTIC ENDOSCOPE. (Eng.) Hopkins, H. H. *In: Modern Topics in Gastrointestinal Endoscopy.* Edited by K. F. R. Schiller and P. R. Salmon. (Chicago: William Heinemann Medical Books): 15-62; 1976.

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1105 EXPERIENCE WITH THE APPLICATION OF A RADIOISOTOPE TECHNIQUE IN THE STUDY OF GASTRIC EMPTYING. (Ita.) Corinaldesi, R.; Luchetta, L.; Putti, C.; Ricci, P.; Casadio, R., Fabbri, R.; *et al.* (Istituto di Gastroenterologia dell'Universita di Bologna, Bologna, Italy). *Boll. Soc. Ital. Biol. Sper.* 52(18):1471-1477; 1976.

1106 STAGES AND LIMITS OF DIAGNOSTICS IN LIVER DISEASES. (Ger.) Martini, G. A. (Medizinische Universitatsklinik, Mannkopffstrasse 1, D-3550 Marburg/Lahn, W. Germany). *Internist (Berlin)* 18(3):149-155; 1977.

1107 STUDY OF HUMORAL IMMUNITY IN HUMAN SCHISTOSOMIASIS. (Fre.) Pinon, J. M.; Dropsy, G. (Hopital de Reims, 51100, Reims, France). *Bull. Soc. Pathol. Exot.* 69(3):238-244; 1976.

1108 PLASMA BINDING, RENAL AND BILIARY EXCRETION OF TWO INTRAVENOUS CHOLANGIOGRAPHIC AGENTS IN NORMAL AND JAUNDICED SUBJECTS [Abstract]. (Eng.) Doran, J.; Vennart, W.; Bell, G. D. (City Hosp., Nottingham, England). *Gut* 18(11):A984; 1977.

1109 ASSESSMENT OF OBSERVER ERROR IN UPPER GASTROINTESTINAL ENDOSCOPY [Abstract]. (Eng.) Holden, R. J.; Crean, G. P. (Southern General Hosp., Glasgow, Scotland). *Gut* 18(11):A941; 1977.

1110 NEUROLEPTANALGESIA VERSUS DIAZEPAM AS A PREMEDICATION FOR UPPER GI TRACT ENDOSCOPY [Abstract]. (Eng.) Birt, C.; Davies, G.; Ashby, B. S. (General Hosp., Southend, England). *Gut* 18(11):A940; 1977.

1111 HEMOCCULT TESTING [Letter to Editor]. (Eng.) Ostrow, J. D.; Fleisher, M.;

Schwartz, M. K.; Winawer, S. J. (Veterans Admin. Hosp., Philadelphia, PA 19104). *Gastroenterology* 73(5):1185-1186; 1977.

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1113 LIVER VOLUME DETERMINATION BY ULTRASOUND: A FEASIBILITY STUDY. (Eng.) Carr, D.; Duncan, J. G.; Railton, R.; Smith, C. B. (Glasgow Royal Infirmary, Glasgow, Scotland). *Br. J. Radiol.* 49(585):776-778; 1976.

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1117 PEROPERATIV [sic] CHOLEDOCHOSCOPY [Abstract]. (Eng.) Rosseland, A. R. (Centralhosp. Akershus, N-1474 Nordbyhagen, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):83; 1977.

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1119 FIBEROPTIC COLONOSCOPY. COMPLICATIONS OF COLONOSCOPY AND POLYPECTOMY. (Eng.) Smith, L. E. (Natl. Naval Medical Center, Bethesda, MD 20014). *Dis. Colon Rectum* 19(5):407-412; 1976.

1120 THE NEW SHORT FLEXIBLE SIGMOIDOSCOPES OF ACMI AND WOLF IN THE ENDOSCOPIC EXAMINATION OF THE RECTUM AND SIGMOID COLON [Abstract]. (Eng.) Otto, P.; Huchzermeyer, H.; Helmstaedt, D. (Kreiskrankenhaus Grossburgwedel, Hannover, W. Germany). *Endoscopy* 9(3):193; 1977.

1121 RELIABILITY OF GASTRIC ANALYSIS IN GASTRO-DUODENAL PATHOLOGY. (Spa.) Meeroff, J. C.; de Larrechea, I. (Hospital Escuela General Jose de San Martin, Buenos Aires, Argentina. *Rev. Esp. Enferm. Apar. Dig.* 48(2):169-186; 1976.

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1127 DIAGNOSIS OF EXOCRINE PANCREATIC INSUFFICIENCY BY MEANS OF A SYNTHETIC PEPTIDE [Letter to Editor]. (Fre.) Leger, L.; Guest, M.; Souciet, C. (Hopital Cochin, 27, rue du Fg. St-Jacques, 75014 Paris Cedex 14, France). *Nouv. Presse Med.* 6(18);1569; 1977.

1128 SCANNING OF THE PANCREAS WITH SELENOMETHIONINE. (Rus.) Bykov, G. A.; Mazaev, A. P.; Nad', M. (A. V. Vishnevskii Inst. Surgery, USSR Acad. Medical Sciences, Moscow, USSR). *Med. Radiol. (Mosk.)* 22(2):87-89; 1977.

1129 SERUM ENZYMOLOGICAL CHANGES FOLLOWING ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY (ERCP). (Hun.) Nemesanszky, E.; Papp, J.; Tulassay, Zs. (Orvostovabbkepzo Intezet, I. Belgyogyaszati Klinika, Budapest, Hungary). *Orv. Hetil.* 118(31):1831-1834; 1977.

1130 ENDOSCOPIC CHOLANGIOPANCREATOGRAPHY: RADIOLOGICAL ASPECTS OF THE NORMAL AND PATHOLOGICAL PANCREAS. (Fre.) Bourgie, J. R.; Bessette, G. (Hopital Maisonneuve-Rosemont, 5415, boulevard de l'Assomption, Montreal HIT 2M4, Canada). *Rev. Fr. Gastroenterol.* (121):20-30; 1976.

1131 DRIP INFUSION CHOLECYSTO-CHOLANGIOGRAPHY WITH IODOXAMATE (BC-17) (III)--ESPECIALLY COMPARED WITH IODIPAMIDE. (Jpn.) Tanimura, H.; Takenaka, M.; Setoyama, M.; Kamata, T. (Kyoto Univ. Sch. Medicine, Sakyo-Ku, Kyoto, 606, Japan) *Arch. Jpn. Chir.* 46(3):313-325; 1977.

1132 INVESTIGATION OF THE RELEVANCE OF CLINICAL CHEMICAL PARAMETERS IN LIVER AND BILE DUCT DISEASES BY MULTIVARIATE DISCRIMINANT ANALYSIS. (Ger.) Nilius, R. (I. Medizinischen Klinik der Martin-Luther-Universitat, Ernst-Grube-Strasse, PSF 63, DDR-401 Halle, E. Germany). *Z. Gesamte Inn. Med.* 32(4):50-54; 1977.

1133 RESPONSE OF HEPARIN-CONTAINING CELLS AND FIBRINOLYTIC SPLIT PRODUCTS TO SULFOBROMOPHTHALEIN OR INDOCYANINE GREEN IN PATIENTS WITH NORMAL AND PATHOLOGICAL LIVER (Ger.) Frick, G.; Frick, U.; Brugmann, E. (Klinik fur Innere Medizin der Wilhelm-Pieck-Universitat, Leninallee 35, 25 Rostock, E. Germany). *Z. Gesamte Inn. Med.* 31(15): 579-583; 1976.

1134 SURGICALLY ACTIVE LAPAROSCOPY AND FIBROCHOLEDOCHOSCOPY IN DIAGNOSIS AND TREATMENT OF BILIARY TRACT DISEASES. (Rus.) Napalkov, P. N.; Arten'eva, N. N.; Kashurin, V. S.; Karchemnik, E. A. (Leningrad Inst. Medicine and Hygiene, Leningrad, USSR). *Vestn. Khir.* 117(11):39-44; 1976.

1135 ON THE GENESIS OF RHEOGRAPHIC WAVES OF THE LIVER. (Rus.) Andreeva, N. Ya. (Fourth Dept., USSR Ministry Public Health, Moscow, USSR). *Ter. Arkh.* 48(9):49-53; 1976.

1136 STANDARDIZED DIRECT IMMUNOFLUORESCENCE. DIFFERENTIATION OF MITOCHONDRIAL, MICROSOMAL AND RIBOSOMAL ANTIBODIES. (Ger.) Storch, W. (Neurologische Klinik, Karl-Marx-Univ., Karl-Marx-Stadter Strasse 50, DDR-7039 Leipzig, E. Germany). *Z. Gesamte Inn. Med.* 32(4):60-63; 1977.

1137 SURVEY OF CLINICAL SIGNIFICANCE OF SERUM BILIRUBIN LEVEL. (Jpn.) Nakaguchi, N. (Nagasaki Univ. Sch. Medicine, Nagasaki, Japan). *Jpn. J. Clin. Pathol.* 24(2):118-124; 1976.

1138 DIAGNOSTIC STRATEGY IN PRIMARY AND METASTATIC NEOPLASMS OF THE LIVER. (Rus.) Izimbergenov, N. I. (Central Inst. Advanced Medical Studies, Moscow, USSR). *Sov. Med.* (2):123-126; 1977.

1139 RENAL CARCINOMA DETECTED BY PERCUTANEOUS LIVER BIOPSY. (Hun.) Kapui, M.; Fedor, I. (Varosi Korhaz, Belgyogyaszati Osztaly, Kazincbarcika, Hungary). *Orv. Hetil.* 118(14):811-812; 1977.

1140 BIOCHEMICAL DIFFERENTIAL DIAGNOSIS OF THE HEPATIC FORM OF GLYCOGENOSIS. (Slo.) Matheova, E.; Pavkovcekova, O.; Fedorova, E.; Tischler, V.; Kahanec, D. (Krajska detska nemocnica, 040 00 Kosice, Czechoslovakia). *Cas. Lek. Cesk.* 115(2/3): 56-60; 1976.

1141 COMPOSITION OF NEUTRAL BLOOD PLASMA LIPIDS IN VIRAL HEPATITIS. (Rus.) Bondarenko, B. N.; Siniak, K. M.; Sokol, A. S.; Orgel, M. Ya. (Kiev Scientific Res. Inst. Epidemiology, Microbiology and Parasitology, Kiev, USSR). *Vrach. Delo* (9): 148-152; 1976.

1142 ASSESSMENT OF THE VALUE OF SELECTED METHODS IN THE DIAGNOSIS OF LIVER DISEASES WITH SPECIAL REFERENCE TO VIRAL HEPATITIS ON THE BASIS OF PERSONAL OBSERVATIONS. (Pol.) Sowa, J. (Klinika Chorob Zakaznych Instytutu Medycyny Wewnetrznej AM, Karkow, Poland). *Pol. Tyg. Lek.* 32(2): 71-72; 1977.

1143 DOUBLE CONTRAST EXAMINATION OF THE SMALL BOWEL. (Eng.) Ekberg, O. (Malmo General Hosp., S-214 01 Malmo, Sweden). *Gastroenterol. Radiol.* 1(4):349-353; 1977.

1144 OESOPHAGEAL ACID PERFUSION TEST AS A COMPLEMENT TO WORK TEST IN PATIENTS WITH CHEST PAIN. (Eng.) Areskog, M.; Tibbling, L.; Wranne, B. (Univ. Hosp., Linkoping, Sweden). *Acta Med. Scand.* 201(6):559-562; 1977.

1145 CLINICAL INFORMATION FROM MEDICAL IMAGES. A COMPARISON OF TWO TYPES OF ULTRASONIC IMAGE IN THE DETECTION OF FOCAL INTRAHEPATIC LESIONS. (Eng.) Robertson, D. A. R.; Evans, A. L.; McCreath, G. (Southern General Hosp., Glasgow, Scotland). *Br. J. Radiol.* 50(595):499-503; 1977.

See also, 0826, 0827, 0902, 0904, 0905, 1038, 1146, 1152, 1153, 1176, 1179, 1185, 1188, 1195, 1231, 1241, 1251, 1269, 1272, 1299, 1302, 1309, 1342, 1344, 1345, 1346, 1348, 1353, 1366, 1368, 1369, 1376, 1384, 1395, 1399, 1402, 1444, 1467, 1476, 1486, 1519, 1529, 1530, 1531, 1539, 1545, 1550, 1558, 1559, 1560, 1561, 1562, 1563, 1578, 1580, 1587, 1589, 1594, 1614, 1622, 1652, 1657, 1693.

GASTROINTESTINAL DISEASES

ESOPHAGUS

1146 INTRODUCTION OF A PNEUMATIC DILATOR WITH THE HELP OF A GASTROSCOPE IN ACHALASIA--A NEW, NONTRAUMATIC TECHNIC. (Eng.) Singal, S. K.; Barfi, R.; Hossain, T. Z. (Allen Park Veterans Admin. Hosp., Detroit, MI). *Am. J. Gastroenterol.* 67(5):494-496; 1977.

A new endoscopic technique to introduce a pneumatic dilator through the lower esophageal sphincter is described in a patient with achalasia. The patient, a 96-yr-old man, was found to have a markedly dilated esophagus with a narrow distal end. An esophageal motility study showed no peristaltic response to swallowing, increased lower esophageal sphincter pressure with no relaxation on swallowing, and a positive response to the urecholine test. A loop of surgical suture (27.5 cm diameter) was tied to the flexible spring-wire tip of a Jackson-Mosher pneumatic dilator before it was passed into the esophagus with the patient lying on his left side. A GIF-K gastroscope was introduced into the esophagus beside the dilator. The loop of silk was identified and caught by biopsy forceps passed through the gastroscope. The forceps were then withdrawn so that their tip lay just inside the end of the biopsy channel. Under direct vision, the gastroscope was passed through the lower esophageal sphincter into the stomach pulling the silk and the dilator along with it. Once the lower end of the pneumatic bag was ascertained to lie below the lower esophageal sphincter area, the biopsy forceps were opened to free the silk loop. The gastroscope was then removed while the pressure was maintained on the stylet to prevent the dilator from being withdrawn. The position of the pneumatic bag was then adjusted fluoroscopically, and a pressure of 6.2 kg/6.5 cm^2 was applied for 2 min. The patient improved markedly after dilatation. This technique can be very useful in patients with achalasia associated with either sigmoid-shaped esophagus or epiphrenic diverticulum where blind introduction of the dilator may not be possible.

1147 LONG-TERM ESOPHAGEAL FUNCTION FOLLOWING REPAIR OF ESOPHAGEAL ATRESIA. (Eng.) Orringer, M. B.; Kirsh, M. M.; Sloan, H. (C7175 Univ. Hosp., Ann Arbor, MI 48109). *Ann. Surg.* 186(4):436-443; 1977.

The long-term esophageal motor disturbances and incidence of gastroesophageal reflux after surgical repair of esophageal atresia were evaluated in 22 patients postoperatively. The patients were evaluated by personal interview and by esophageal manometry and acid reflux testing. Previous barium swallow examinations had demonstrated varying degrees of anastomotic narrowing (12 patients), abnormal esophageal motor function (11), gastroesophageal reflux (2), and hiatal hernia (1). Ten patients experienced intermittent dysphagia for solid foods. Seven had typical symptoms of gastroesophageal reflux. Esophageal function tests, including manometry and intraesophageal pH recording, demonstrated varying abnormalities of esophageal motility in 21 patients and moderate to severe gastroesophageal reflux in 13. Two patients required reconstruction of the esophagogastric junction for control of severe reflux esophagitis. The unexpected high incidence of gastroesophageal reflux in these patients, coupled with abnormal esophageal motility that impairs normal acid clearing, renders them more prone to reflux esophagitis. Careful long-term evaluation for gastroesophageal reflux and its complications is indicated following primary repair of esophageal atresia. Evaluation of esophageal function with intraesophageal pressure and pH recordings is a far more sensitive indicator of esophageal physiology than the barium swallow examination.

1148 ROLE OF LOWER ESOPHAGEAL SPHINCTER INCOMPETENCE IN RECURRENT PNEUMONIA AFTER REPAIR OF ESOPHAGEAL ATRESIA. (Eng.) Whitington, P. F.; Shermeta, D. W.; Seto, D. S. Y.; Jones, L.; Hendrix, T. R. (Univ. Wisconsin Sch. Medicine, Madison, WI 53706). *J. Pediatr.* 91(4):550-554; 1977.

To define the etiology of recurrent aspiration pneumonitis after the successful repair of esophageal atresia (EA), esophageal manometry was performed in eight patients (aged 11 weeks to 20 yr) who had undergone repair of EA and tracheoesophageal fistula. Two patients who had had recurrent pneumonia had subnormal lower esophageal sphincter pressure; they also had a history of severe regurgitation, and a barium esophagram demonstrated free gastroesophageal reflux. The lower esophageal sphincter incompetence in these patients was apparently corrected by the administration of bethanechol. These studies show that recurrent pneumonitis after repair of EA is often due to an incompetent lower esophageal sphincter with reflux and aspiration.

1149 PHARMACOTHERAPY OF AN ULCER IN BARRETT'S ESOPHAGUS: CARBENOXALONE AND CIMETIDINE. (Eng.) Thompson, W. G.; Barr, R. (Ottawa Civic Hosp., 1053 Carling Ave., Ottawa, Ontario K1Y 4E9, Canada). *Gastroenterology* 73(4):808-810; 1977.

The case of a 70-yr-old man who had specialized columnar epithelium in the esophagus up to the upper esophageal sphincter, 19 cm from the incisor teeth, is presented. The patient had a small sliding hiatal hernia, but the lower esophageal sphincter was located at 39-41 cm. Above the sphincter, the patient had large peptic ulcers that failed to heal after 1 yr of a conventional antireflux regimen. Carbenoxalone sodium (50 or 100 mg, t.i.d.) led to healing in 3 months. However, the ulcers recurred after discontinuation of the drug. On a second attempt, carbenoxalone effected modest healing, but

was stopped because of hypertension and hypokalemia. Cimetidine therapy (300 mg, t.i.d. and 600 mg at bedtime) led to complete healing of the ulcer within a month. The authors conclude that both carbenoxalone and cimetidine are useful in the treatment of an ulcer in Barrett's esophagus. However, cimetidine may be more rapidly effective and does not produce dangerous side-effects.

1150 MANAGEMENT OF COMPLICATIONS OF FUNDOPLICATION AND BARRETT'S ESOPHAGUS. (Eng.) Burnett, H. F.; Read, R. C.; Morris, W. D.; Campbell G. S. (Veterans Admin. Hosp., 300 E. Roosevelt Rd., Little Rock, AR 72206). *Surgery* 82(4):521-530; 1977.

Experience with patients requiring reoperation for complications of fundoplication and with the management of columnar-lined distal esophagus (Barrett's) is reviewed. Fifteen patients required reoperation for gastric fistula (6), progressive esophagitis/stricture (4), obstruction by the wrap (3), carcinoma (1), failed pericardioesophagoplasty (1), and gastric ulcer (1). Of the six patients with gastric fistulas, five had transthoracic Nissen procedures, and three died without surgical closure having been attempted. One survived after operative closure, and two survived after esophageal exclusion. Progressive esophagitis/stricture followed abdominal Nissen operations in two patients, Belsey procedure (1 case), and Belsey with Collis gastroplasty (1 case). Remedial thoracic Nissen operations gave good results in two patients. Esophageal obstruction by the wrap in three patients was managed by resection and distal colon interposition, with good results. Those with cancer, a failed pericardioesophagogastrectomy, Woodward procedure, and wedge resection, resp., and all are doing well. Eight patients had Barrett's esophagus, one of whom had an associated cancer. Fundoplication gave good results in three of five patients; there was one death from gastric fistula. Failed operations on the gastroesophageal junction can be rendered by carefully selected surgical techniques based on a thorough preoperative evaluation of the patient. Barrett's esophagus is managed successfully with fundoplication and dilatation. There is an increased incidence of cancer in this disorder, which demands long-term follow-up.

1151 EXPERIENCE IN THE MANAGEMENT OF ACUTE ESOPHAGEAL BURN. (Ger.) Muller, H. (Ful-, Orr-, Es Bronchologiai Osztaly, Magyei Korhaz, 6601 Szentes, Hungary). *HNO* 25(5):168-171; 1977.

The treatment of 151 patients with acute esophageal burns caused by corrosive substances is described. The patients included 4 2- to 4-yr-old children, and 23 patients in severe, critical condition. Pain relief, administration of atropine and pipolphenin injection are necessary at first, but gastric lavage is not performed, and the poison is not neutralized. The principal treatment consists of cortisone administration and mechanical dilatation of the esophagus to prevent the formation of granulation tissue and esophageal stricture. Blind bougienage is contraindicated because of its inherent hazards. Cortisone is administered to adults in daily doses of 200-400 mg, and the dose is reduced by 50-100 mg after 5-8 days. Children (up to age 10) receive 100-200 mg/day, depending on age. After cessation of dysphagia, cortisone is replaced by prednisolone, which is given in daily doses of 20-30 mg for maintenance. Antibiotic therapy with penicillin (3-5 million U for adults) is absolutely necessary during cortisone therapy. All patients with slight to medium degree burns were healed by cortisone therapy, but 6 of the 23 patients with severe burns died (5 from uncontrollable bleeding, 1 from perforation of the trachea). Gastric perforation and gastric ulcer as complications of cortisone treatment were not observed. The findings indicate that cortisone is of great value in the treatment of severe esophageal burns.

1152 THE ROLES OF ESOPHAGOSCOPY VS. RADIOGRAPHY IN DIAGNOSING BENIGN PEPTIC ESOPHAGEAL STRICTURES. (Eng.) Hiatt, G. A. (Fronk Clinic, 839 South Beretania St., Honolulu, Hawaii 96813). *Gastrointest. Endosc.* 23(4):194-195; 1977.

The correlation between endoscopic and radiographic diagnosis of benign peptic esophageal stricture was studied retrospectively in 52 patients found to have significant strictures at esophagoscopy. Radiographs read as positive for stricture ranged from 40% to 34.1% when taken 1-8 months before endoscopy. None of 10 patients who had a barium swallow after the endoscopic diagnosis was reported as having a stricture. These data indicate that radiography is not entirely reliable in the diagnosis of benign peptic esophageal strictures and that esophagoscopy should be routinely employed when dysphagia is a symptom.

1153 REMARKABLY LARGE, BENIGN ESOPHAGEAL TUMOR. DIFFICULTIES IN DIAGNOSIS. (Eng.) Orchard, J. L.; Peternel, W. W.; Arena, S. (Mercy Hosp., Pittsburgh, PA 15219). *Am. J. Dig. Dis.* 22(3):266-269; 1977.

The case of a patient with a leiomyoma of the esophagus is reported because the tumor reached extraordinary size before becoming symptomatic and caused some difficulty in diagnosis despite its large size. The diagnosis was made with the fiberoptic esophagoscope after standard roentgenograms and rigid esophagoscopy were inconclusive. The tumor was removed through a cervical incision. Attention is called to the potential fatal outcome of these lesions.

1154 ESOPHAGEAL ULCERS IN IMMUNODEFICIENCY WITH ELEVATED LEVELS OF IgM AND NEUTROPENIA. (Eng.) Vanderhoff, J. A.; Rich, K. C.; Stiehm, E. R.; Ament, M. E. (Dept. Pediatrics, MDCC 22-442 UCLA, Center Health Sciences, Los Angeles, CA 90024). *Am. J. Dis. Child.* 131(5):551-552; 1977.

The case of a 17-yr-old boy with immunodeficiency, elevated levels of immunoglobulin M, and neutropenia who developed distal esophageal ulcers and stricture is presented. Although his lower esophageal sphincter pressure was decreased, he had normal acid reflux test results, normal findings from acid clearing studies, and absence of diffuse esophagitis at esophagoscopy. Neutropenia and hypogammaglobulinemia are postulated to be pathogenic factors in his esophageal ulcers.

1155 MINUTE EARLY CARCINOMA OF THE ESOPHAGOGASTRIC JUNCTION. (Jpn.) Suzuki, S.; Nagayo, T.; Murakami, H.; Takemoto, T. (Tokyo Women's Medical Coll., Tokyo, Japan). *Stomach Intest. (Tokyo)* 11(6):697-704; 1976.

1156 MANAGEMENT OF INFANTS WITH DIAPHRAGMATIC HERNIA. (Eng.) Collins, D. L.; Marks, L.; Edwards, D.; Kirkpatrick, S. E.; Nyhan, W. L. (Univ. California San Diego Sch. Medicine, San Diego, CA 92103). *West. J. Med.* 127(6):479-486; 1977.

1157 EXPERIENCES WITH CONTINUOUS GASTROSTOMY [Abstract]. (Eng./Ger.) Klinke, F.; Heiseke, E.; Wedell, J. (Kreiskrankenhaus Herford, D-4900 Herford, W. Germany). *Langenbecks Arch. Chir.* 345:599; 1977.

1158 CRITERIA FOR OPERATIVE TECHNIQUES FOR AVOIDANCE OF SUTURE INSUFFICIENCY WITH ESOPHAGOGASTROSTOMY [Abstract]. (Eng./Ger.) Kunath, U.; Stelzner, F. (Chirurgische Universitatsklinik, D-5300 Bonn-Venusberg, W. Germany). *Langenbecks Arch. Chir.* 345:599-600; 1977.

1159 COLONIC REPLACEMENT OF THE OESOPHAGUS [Abstract]. (Eng.) Roed-Petersen, K.; Pedersen, S. A.; Madsen, C. M. (Odense Univ. Hosp., Odense, Denmark). *Scand. J. Gastroenterol.* 12 (Suppl. 45):78; 1977.

1160 FILM SHOWING COLONIC REPLACEMENT OF THE OESOPHAGUS [Abstract]. (Eng.) Madsen, C. M. (Odense Univ. Hosp., Odense, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):59; 1977.

1161 DISORDERS OF ESOPHAGEAL MOTILITY. (Ger.) Koelz, H. R.; Lepsien, G. (Stadtspital Triemli, Medizinische Klinik, Birmensdorfer Str. 497, CH-8063 Zurich, Switzerland). *Z. Gastroenterol.* 15(3):167-174; 1977.

1162 CURRENT THERAPY OF ESOPHAGEAL ATRESIA [Abstract]. (Ger.) Hollwarth, M.; Sauer, H. (No affiliation given). *Wien. Med. Wochenschr.* 127 (15):494; 1977.

1163 ANASTOMOTIC LEAKAGE AFTER CURE OF ESOPHAGEAL ATRESIA. (Fre.) Revillon, Y.; Bondeux, D.; Lavaud, J.; Fekete, C. M.; Cloup, M.; Pellerin, D. (Hopital des Enfants Malades, 149, rue de Sevres, 75015 Paris, France). *Ann. Chir. Infant.* 18(3):209-217; 1977.

1164 TOTAL PARENTERAL ALIMENTATION (TPA) WITH A COMBINATION OF CARBOHYDRATES FOR SURGICAL PATIENTS WITH CARCINOMA OF THE ESOPHAGUS. (Chi.) Wang, P. Y.; Hsu, T. L.; Chien, K. Y.; Lu, K. S.; Meng, H. C. (Veterans General Hosp., Taipei, Taiwan). *Chi. Med. J.* [*Engl.*] (22):144-159; 1975.

1165 RADICALNESS OF SURGERY FOR ESOPHAGEAL CANCER. (Rus.) Shanin, V. E. (Khimki Central Regional Hosp., Moscow Oblast, Khimki, USSR). *Klin. Khir.* (3):65-66; 1977.

1166 COMBINED BENIGN AND MALIGNANT TUMORS OF THE ESOPHAGUS. (Ger.) Farschidpour, D.; Nadji, A. (Krankenhauses Farabi, Universitat Tehran, Tehran, Iran). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 126(3):272; 1977.

1167 REPORT OF A CASE OF GIANT EPIPHRENIC DIVERTICULUM. (Spa.) Gasco Benaque, J. L.; De Pablo Redondo, L. (Residencia Sanitaria "Virgen de la Salud," Toledo, Spain). *Rev. Esp. Enferm. Apar. Dig.* 50(4):463-468; 1977.

1168 ESOPHAGEAL LOCALIZATION OF FOLLICULAR DYSKERATOSIS (DARIER'S SYNDROME). RADIOLOGICAL ASPECTS. REPORT OF FOUR CASES. (Fre.) Larregue, M.; Katz, M.; Gasquet, C.; Lansmann, M.; Bressieux, J. M. (Service de Dermatologie, Hotel-Dieu, F 86000 Poitiers, France). *Ann. Med. Interne* 128(5):487-491; 1977.

1169 MORPHOLOGICAL CHANGES IN GASTRIC MUCOSA IN CHRONIC ESOPHAGITIS. (Rus.) Alexandrova, N. M.; Kolycheva, N. I.; Dril, L. Ya. (Scientific Res. Inst. Oncology Radiology, Kazakh SSR Ministry Public Health, Alma-Ata, USSR). *Klin. Med.* 55(4): 36-39; 1977.

1170 SLIDING HIATAL HERNIA. (Ita.) Montorsi, W. (Ospedale Maggiore "Policlinco," Pad. Beretta Est, Via F. Sforza, 35, 20135 Milano, Italy). *Minerva Med.* 68(1):1-12; 1977.

1171 EVALUATION OF THE INFRACARDIAC BURSA IN THE PATHOGENESIS OF SLIDING HIATAL HERNIA. (Ger.) Kunath, U. (Zentrum der Chirurgie, Johann Wolfgang Goethe-Universitat, Theodor-Stern-Kai 7, D-6000 Frankfurt am Main, W. Germany). *Langenbecks Arch. Chir.* 343(2):161-172; 1977.

1172 REFLUX ESOPHAGITIS OF ARTIFICIAL ESOPHAGUS RECONSTRUCTED FROM THE LARGE INTESTINE IN CHILDREN. (Rus.) Shapiro, G. D. (Novosibirsk Oblast Clinical Hosp., Novosibirsk, USSR). *Vestn. Khir.* 118(2):82-83; 1977.

1173 GASTROESOPHAGEAL REFLUX DISEASE. (Ger.) Guller, R. (Medizinische Universitats-Poliklinik, Hebelstrasse 1, CH-4056 Basel, Switzerland). *Schweiz. Med. Wochenschr.* 107(18):623-626; 1977.

1174 THE Z BAND. ENDOSCOPIC AND HISTOLOGICAL ASPECTS. (Ger.) Clemencon, G.; Gloor, F. (Frohheimweg 12, CH-4600 Olten, Switzerland). *Z. Gastroenterol.* 14(7):704-705; 1976.

See also, 0819, 1082, 1090, 1109, 1144, 1232, 1262, 1393, 1592, 1652, 1657.

STOMACH

1175 CARBOHYDRATE CONTENT OF ENDOSCOPIC GASTRIC BIOPSIES IN CARCINOMA OF THE STOMACH. (Eng.) Machado, G.; Clamp, J. R.; Read, A. E. (Bristol Royal Infirmary, Bristol, England). *Gut* 18(8):670-672; 1977.

Mucous glycoproteins were studied in gastric biopsies of 37 normal controls and in the cancerous and normal biopsies of 27 patients with gastric carcinoma. There were statistically significant ($p<0.001$) changes in mannose and *N*-acetylgalactosamine levels in normal and carcinomatous stomachs in both secretors and nonsecretors. The mean mannose level in the 25 normal secretors was 1.0 (residue of mannose relative to a galactose content of 6.0 residues). Among the 12 cancer patients who were secretors, the mean mannose levels in the cancerous and noncancerous areas were 1.7 and 1.5, resp. In the nonsecretors, the corresponding mannose contents were 1.3, 2.2, and 2.0, resp. The levels of *N*-acetylgalactosamine in the secretors were 4.0, 2.6, and 2.8, resp., while in the nonsecretors they were 3.8, 2.2, and 2.6, resp. These studies show that there is a significant increase in mannose content and a significant decrease in *N*-acetylgalactosamine in the gastric mucus of patients with gastric cancer, in comparison with the levels in normal subjects. The fact that both the cancerous and noncancerous areas of cancer-bearing stomach show carbohydrate changes suggests a change in the relative amounts of these two fractions and therefore a corresponding change in the cells giving rise to them. Carbohydrate analysis of adherent gastric mucus could be useful in detecting susceptibility to gastric cancer.

1176 OUTCOME OF GASTRIC CARCINOMA DETECTED BY GASTRIC MASS SURVEY IN JAPAN. (Eng.) Kaneko, E.; Nakamura, T.; Umeda, N.; Fujino, M.; Niwa, H. (Sch. Medicine, Hamamatsu Univ., Hamamatsu Japan). *Gut* 18(8):626-630; 1977.

To determine the value of the gastrocamera in mass screening for gastric carcinoma, the results of six selected mass survey groups consisting of 90,557 subjects were studied. One hundred thirty seven cases of gastric carcinoma were detected and followed up postoperatively for up to 16 yr. They consisted of 55 cases of early and 74 cases of advanced carcinoma; the stage of malignancy was unknown in the remaining cases. The relative 5-yr and 10-yr survival rates in the 137 cases were 0.628 and 0.642, resp., and those for early carcinoma were 0.959 and 1.016, resp. These rates are remarkably high. The 5-yr survival rate in those who were followed up after operation for more than 5 yr (83 cases) was 0.554, which is much higher than that of outpatients who presented only after developing symptoms. The use of the gastrocamera markedly improved the detection rate of gastric carcinoma in mass screening programs.

1177 CELL-MEDIATED IMMUNITY IN PATIENTS WITH GASTRIC CARCINOMA: CORRELATION BETWEEN CLINICAL STAGE AND IMMUNOCOMPETENCE. (Eng.) Branco, P. D.; Raia, A. A.; Varella, A. D.; Kopersztich, S.; Abrao, S.; Nakaie, M.; Castro, J. P. (Facultade de Medicina, Universidade de Sao Paulo, Sao Paulo, Brazil). *Chir. Gastroenterol.* 11(2): 187-191; 1977.

To determine whether defective cell-mediated immunity (CMI) can predispose to the establishment and progression of neoplasms, CMI was investigated in 75 patients who had undergone surgery for gastric carcinomas. The 2,4-dinitrochlorobenzene (DNCB) sensitization test was used as an index of CMI. The patients were divided into three groups according to the stage of the tumor: patients with localized and resectable tumors (LD); patients with disseminated tumors, but with the progression of the disease restricted to the regional lymph nodes (DD), in whom it was possible to achieve some surgical procedure; and patients with metastatic disease with great extension of the primary disease and with widespread metastasis (MD). In the 27 patients with LD, 17 negative and 10 positive DNCB reactions (62.96% and 37.04%, resp.) were observed. In 26 patients with DD, 17 negative (65.38%) and 9 positive reactions (34.62%) were seen. In 22 patients with MD, 20 negative (90.9%) and 2 positive reactions (9.1%) were observed. During a 6-month follow-up, 20 DNCB-negative patients died, while only 4 DNCB-positive patients died. The results demonstrate

a marked difference in CMI between patients with MD, DD, and LD.

1178 GASTRIC MUCOSAL BIOPSY IN EOSINOPHILIC (ALLERGIC) GASTROENTERITIS. (Eng.) Katz, A. J.; Goldman, H.; Grand, R. J. (Children's Hosp. Medical Center, Boston, MA 02115). *Gastroenterology* 73(4):705-709; 1977.

Six patients were studied to assess the value of gastric mucosal biopsy in establishing the diagnosis of eosinophilic (allergic) gastroenteritis. The gastric mucosa was abnormal in all cases, revealing marked eosinophilic infiltration and necrosis and regeneration of the epithelial cells. A single gastric biopsy was diagnostic in all six cases and provided the primary diagnosis in three. Intestinal biopsies varied considerably in appearance even in the same patient, ranging from normal to villous shortening and crypt hyperplasia with epithelial cell necrosis. Other patients with peripheral eosinophilia alone or intestinal disease of other etiologies had normal gastric biopsies. Gastric mucosal biopsy appears to be valuable in the diagnosis of eosinophilic gastroenteritis and should be routinely obtained. The gastric lesion may contribute to the protein loss, gastrointestinal bleeding, and iron-deficiency anemia observed in this condition.

1179 HEMOLYTIC SUBSTANCES IN THE GASTRIC CONTENTS: FREQUENCY OF LYSOLECITHIN DETECTION. (Ger.) Clemencon, G.; Burgi, W. (Kantonsspital Olten, CH-4600 Olten, Switzerland). *Schweiz. Med. Wochenschr.* 107(27):952-956; 1977.

Samples of gastric contents were taken from 152 patients with gastroduodenal reflux after an overnight fast and examined for hemolytic activity and lysolecithin content. The hemolysis tests were positive in 97/152 cases: in 13/29 patients with normal mucosa, in 13/20 patients with reflux esophagitis, in 33/49 patients with gastric ulcer, in 10/12 patients with gastric erosion, in 15/25 patients with superficial gastritis, and in 13/17 patients with chronic atrophic gastritis with intestinal metaplasia. Eighty-eight of the positive samples were analyzed by thin-layer chromatography for lysolecithin content. Lysolecithin was not found in nine samples. The lysolecithin levels averaged 41.5 mg/100 ml in 13 patients with normal gastric mucosa, 28.5 mg/100 ml in 9 patients with reflux esophagitis, 19.7 mg/100 ml in 26 patients with gastric ulcer, 38.4 mg/100 ml in 8 patients with gastric erosion, 61.3 mg/100 ml in 13 patients with superficial gastritis, and 43.6 mg/100 ml in 10 patients with chronic atrophic gastritis with intestinal metaplasia. The lysolecithin levels found in patients without gastroduodenal reflux in previous investigations averaged 0.9 mg/100 ml. The pH value of the gastric contents, ranged from 1.1 to 7.8 and did not correlate with the lysolecithin level or the result of the hemolysis test, nor was there any correlation between the lysolecithin level and bilirubin level. Along with data in the literature, these findings indicate that lysolecithin may exert a pathogenic effect in the development of various gastric lesions.

1180 LOCATION OF ULCER IN THE STOMACH AND PYLOROANTRAL HYPERTROPHY: CORRELATION BETWEEN THE SITE OF ULCER AND GASTRIC WALL HYPERTROPHY IN GASTRIC ULCER. (Ger.) Liebermann-Meffert, D.; Allgower, M. (Chirurg. Dept., Kantonsspital Basel, CH-4004 Basel, Switzerland). *Z. Gastroenterol.* 14(6):613-619; 1976.

Relationships between the localization of gastric ulcer and hypertrophy of the pyloroantral wall (musculature + submucosa + mucosa) were studied in a random series of 77 resected specimens with solitary (60) and multiple (40) gastric ulcers. Sixty-three ulcers were localized to the lesser curvature, 29 to the posterior wall, 6 to the anterior wall, and 2 to the greater curvature. No difference was found between solitary and multiple ulcers in terms of their localization, but ulcers localized to the posterior wall were more frequent in men than in women. Most of the 100 ulcers were at distances of up to 90 mm from the pylorus. The distance between solitary ulcers and the pylorus was usually 20-60 mm, that between multiple ulcers and the pylorus, 0-40 mm. The distance was about 60 mm in most cases, with some difference between men and women in this respect. No significant correlation was found between the distance of solitary and multiple ulcers from the pylorus and the hypertrophy of the entire pyloroantral wall, but the musculature was significantly less hypertrophied in the case of solitary ulcers further from the pylorus, and significantly more hypertrophied in the case of multiple ulcers further from the pylorus compared to the cases of ulcers closer to the pylorus ($p<0.02$).

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1186 "EARLY GASTRIC CARCINOMA:" BEGINNING GASTRIC CANCER? SUPERFICIAL CANCER OF THE STOMACH? THE PROBLEM OF ITS EARLY DETECTION. (Fre.) Potet, F. (Hopital Beaujon, F 92118 Clichy Cedex, France). *Gastroenterol. Clin. Biol.* 1(4):313-318; 1977.

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1200 PRIMARY GASTRIC FORMS OF HODGKIN'S DISEASE. (Fre.) Leymarios, J.; Martin, E.; Marche, Cl.; Vissuzaine, Ch.; Debray, Ch. (Hopital Bichat, 170, boulevard Ney, F 75877 Paris Cedex 18, France). *Ann. Med. Interne (Paris)* 128(6/7):549-551; 1977.

See also, 0854, 0861, 0862, 1020, 1057, 1060, 1067, 1068, 1079, 1082, 1090, 1096, 1102, 1109, 1121, 1122, 1123, 1124, 1125, 1150, 1155, 1157, 1158, 1169, 1202, 1203, 1205, 1206, 1208, 1210, 1212, 1214, 1217, 1219, 1220, 1221, 1250, 1253, 1605, 1622, 1633, 1645, 1652, 1657.

STOMACH

Peptic Ulcers

1201 PERFORATION OF CHRONIC PEPTIC ULCERS AFTER CIMETIDINE. (Eng.) Wallace, W. A.; Orr, C. M. E.; Bearn, A. R. (Queen Elizabeth Hosp., Sheriff Hill, Gateshead NE9 6SX, England). *Br. Med. J.* 2(6091):865-866; 1977.

Three cases of chronic peptic ulcer perforation in which abrupt cessation of cimetidine treatment

may have been the cause are reported. Perforation of a duodenal ulcer in the first patient occurred 5 days after he had completed a 6-week course of cimetidine (200 mg thrice daily plus 400 mg at night). The second patient's perforation occurred 10 days after he had completed a 4-week course of cimetidine (200 mg thrice daily plus 400 mg at night) for gastric ulcer, and perforation in the third patient occurred 10 days after she had completed a 6-week course (same dose regimen) for duodenal ulcer. The treatment resulted in relief of symptoms in the two patients with duodenal ulcer but not in the patient with gastric ulcer. All three patients were prescribed cimetidine by their general practitioners and had no pretreatment investigation and no subsequent investigation planned to confirm ulcer healing; the possibility that gastric cancer was present was therefore not excluded. In all patients, the large size of the perforation resulted in moderate to gross contamination of the peritoneal cavity, which precluded definitive ulcer surgery in two patients. The perforations may have occurred because of rebound hyperacidity after cimetidine had been stopped or because the partially healed chronic ulcer, which had responded to cimetidine, may have been less resistant to perforation when exposed to the normal acid concentrations after cimetidine had been stopped.

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(Rus.) Kostromin, A. S.; Sayenko, V. F.; Silchenko, V. P. (Kiev Scientific Res. Inst. Clinical and Experimental Surgery, Kiev, USSR). *Klin. Khir.* (4):49-53; 1977.

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1221 EARLY GASTRIC ULCER AFTER HYPERSELECTIVE VAGOTOMY FOR DUODENAL ULCER. (Fre.) Cuilleret, J.; Etaix, J. P.; Picq, P.; Barthelemy, C.; Colas, M.; Fraisse, H. (C.H.U. Saint-Etienne, 30, rue de Ferdinand Cambon, F 42100 Saint-Etienne, France). *Nouv. Presse Med.* 6(21):1843-1845; 1977.

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See also, 0852, 1105, 1124, 1630, 1652.

SMALL INTESTINE

1223 INFLUENCE OF SMALL INTESTINAL SHUNT AND SMALL INTESTINAL RESECTION ON GASTRIN RELEASE. (Ger.) Wedell, J.; Fritsch, W. P.; Hausamen, T. U. (Chirurg. Abteilung, Kreiskrankenhauses Herford, 4900 Herford, W. Germany). *Z. Gastroenterol.* 14(6):620-625; 1976.

Gastric acid secretion and serum gastrin levels were studied in 10 normal subjects with normal body weight (group 1), in 7 patients who had undergone jejunoileal bypass surgery for excessive obesity 14 months earlier (group 2), and in 3 patients (group 3) who had undergone subtotal small intestinal resection for infarction 1-36 months earlier. Normal gastric acid secretion was found in groups 1 and 2, and the secretion was at the lower limit of the normal range in group 3. There was no significant difference in the fasting serum gastrin levels between groups 1 and 2, but significantly increased fasting serum gastrin levels were found in group 3. Increase in the serum gastrin level was seen in group 1 during the first 2 hr following the administration of a test meal (100 g of homogenized, low-fat raw meat), but no significant change was seen in group 2 (the serum gastrin level showed no change in 3 patients, and marked gastrin release was seen in the other 4). The serum gastrin level was significantly higher in group 3 than it was in group 1 ($p<0.0005$). The hypergastrinemia seen in group 3 after the test meal may be due to the reduced breakdown of gastrin.

1224 THE IMMUNOLOGIC SYSTEM, IMMUNOLOGIC DEFICIENCY DISORDERS AND LYMPHOID HYPERPLASIA OF THE SMALL INTESTINE. (Eng.) Koch, C. T. (Univ. Hosp., Leiden, Netherlands). *Radiol. Clin. (Basel)* 45(2/4):165-174; 1976.

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1232 DISEASES OF GENITO-URINARY TRACT ASSOCIATED TO MALFORMATION OF THE UPPER DIGESTIVE TRACT AND EXTRA-HEPATIC BILIARY TRACT [Abstract]. (Eng.) Goldraich, N. P.; Kilpp, J. C.; Koff, W.; Pranke, M. L.; Montenegro, T. S. (No affiliation given). *Arq. Gastroenterol.* 14(3):162; 1977.

1233 NATAL TEETH, PATENT DUCTUS ARTERIOSUS AND INTESTINAL PSEUDO OBSTRUCTION: A LETHAL SYNDROME IN THE NEWBORN. (Eng.) Harris, D. J.; Ashcraft, K. W.; Beatty, E. C.; *et al.* (Children's Mercy Hosp., Kansas City, MO). *Clin. Genet.* 9(5):479-482; 1976.

1234 RAPID HPLC ANALYSIS OF CONJUGATED BILE ACIDS FROM HUMAN BILE [Abstract]. (Eng.) Shaw, R.; Smith, J. A.; Elliott, W. H. (St. Louis Univ. Sch. Medicine, St. Louis, MO 63104). *Gastroenterology* 73(5):1246; 1977.

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1244 INTUSSUSCEPTION IN SCHONLEIN-HENOCH PURPURA. (Ita.) Violante, N.; De Vanna, C.; Falcone, G. (Istituto di Clinica Pediatrica dell' Universita di Bari, Bari, Italy). *Minerva Pediatr.* 29(9):633-635; 1977.

1245 TREATMENT OF ACUTE COMMISSURAL ILEUS. (Rus.) Zakharova, G. N.; Myshkin, K. I.; Frankfurt, L. A.; Gorfinkel, I. V. (Saratov Medical Inst., Saratov, USSR). *Sov. Med.* (3):69-73; 1977.

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W.; Schmitz-Moormann, P.; Dombrowski, H. (Medizinischen Universitatspoliklinik, Emil-Mannkopff-Strasse, D-3550 Marburg, W. Germany). *Z. Gastroenterol.* 15(5):300-310; 1977.

1248 POLYPS OF LARGE DUODENAL PAPILLA. (Rus.) Vinogradov, V. V.; Bazilevich, F. V. (Dept. Surgery, Patrice Lumumba Univ., Moscow, USSR). *Vestn. Khir.* 118(3):46-50; 1977.

1249 ENDOSCOPIC SPLITTING OF STENOSIS AT THE URETER-ILEUM ANASTOMOSIS IN ILEUM CONDUIT. REPORT OF A CASE. (Ger.) Leisinger, H. J.; Deyhle, P. (Urologische Universitatsklinik, Kantonsspital Zurich, Ramistrasse 100, CH-8091 Zurich, Switzerland). *Helv. Chir. Acta* 44(3):397-401; 1977.

1250 SEVERE DILATION OF THE STOMACH AND DUODENUM AS A CONSEQUENCE OF INBORN SMALL INTESTINAL STENOSIS IN A 13-YEAR-OLD CHILD. (Ger.) Harzheim, I. (Kinderradiologie, Radiologische Klinik Cottbus, DDR-75 Cottbus, E. Germany). *Radiol. Diagn. (Berl.)* 18(2):185-186; 1977.

1251 X-RAY DIAGNOSIS OF COMPLICATIONS FOLLOWING RESECTION OF THE SMALL INTESTINE. (Rus.) Sipukhin, Ya. M.; Baranova, A. E. (Vladivostok Medical Inst., Vladivostok, USSR). *Vestn. Rentgenol. Radiol.* (2):25-32; 1977.

1252 WATER AND SODIUM TRANSPORT IN ALCOHOLICS [Letter to Editor]. (Eng.) Halsted, C. H.; Mekhjian, H. S. (Sch. Medicine, TB 171, Univ. California, Davis, CA 95616). *Gastroenterology* 73(5):1187-1188; 1977.

1253 FUNCTIONAL AND MORPHOLOGIC EXAMINATION OF THE JEJUNAL REPLACEMENT STOMACH [Abstract]. (Eng./Ger.) Brunner, H.; Kempf, P. (Chirurgische Universitatsklinik, Langenbeckstrasse 1, D-6500 Mainz, W. Germany). *Langenbecks Arch. Chir.* 345:595-596; 1977.

1254 CHANGES IN GUT-RELATED HORMONES IN OBESE PATIENTS DURING WEIGHT LOSS INDUCED BY DIET OR BY INTESTINAL BYPASS [Abstract]. (Eng.) Dahms, W. T.; Atkinson, R. L.; Frame, C.; Taylor, I.; Bray, G. A. (Univ. California at Los Angeles, Sch. Medicine, Los Angeles, CA). *Clin. Res.* 26(2):109A; 1978.

1255 LESIONS ASSOCIATED WITH PEUTZ-JEGHERS SYNDROME AND SO-CALLED FAMILIAL POLYPOSIS COLI [Abstract]. (Eng.) Ushio, K. (Natl. Cancer Center Hosp., Tokyo, Japan). *Gastroenterol. Jpn.* 12(4):330; 1977.

1256 EARLY DIAGNOSIS OF BOWEL AND PANCREATIC INJURIES IN BLUNT ABDOMINAL TRAUMA [Abstract]. (Eng./Ger.) Ruckert, K.; Loth, R. (Chirurgische Universitatsklinik, Langenbeckstr. 1, D-6500 Mainz, W. Germany). *Langenbecks Arch. Chir.* 345:586; 1977.

1257 METRONIDAZOLE IN THE TREATMENT OF BYPASS ENTERITIS [Abstract]. (Eng.) Solhaug, J. H. (Hosp. Torsby, Torsby, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):100; 1977.

1258 SUPERIOR MESENTERIC ARTERY SYNDROME. A REPORT ON FOUR OPERATED CASES [Abstract]. (Eng.) Lukes, P.; Rolny, P.; Nilson, A. E.; Gamklou, R.; Darle, N.; Dotevall, G. (Sahlgren Hosp., Univ. Gothenburg, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):57; 1977.

1259 DIET AND INTESTINAL DISEASE [Abstract]. (Eng.) Jones, F. A. (St. Mark's Hosp., London, England). *Nutr. Rep. Int.* 16(4):530; 1977.

1260 MORPHOLOGICAL AND CLINICAL FEATURES OF LATE ONSET LIVER DISEASE FOLLOWING JEJUNO-ILEAL BYPASS FOR OBESITY [Abstract]. (Eng.) Scott, L. D. (Univ. Texas Medical Sch., Houston, TX). *Gastroenterology* 73(5):1245; 1977.

See also, 1044, 1050, 1072, 1074, 1080, 1082, 1083, 1085, 1086, 1088, 1090, 1104, 1109, 1121, 1123, 1143, 1202, 1206, 1216, 1220, 1221, 1264, 1268, 1270, 1271, 1298, 1317, 1321, 1322, 1339, 1359, 1567, 1591, 1605, 1610, 1621, 1629, 1632, 1633, 1638, 1643, 1645, 1649, 1652, 1657, 1658, 1661, 1662, 1665, 1673.

SMALL INTESTINE

Malabsorption

1261 IMMUNOGLOBULINS IN JEJUNAL MUCOSA AND SERUM FROM PATIENTS WITH ADULT COELIAC DISEASE. (Eng.) Baklien, K.; Brandtzaeg, P.; Fausa, O. (Rikshospitalet, Oslo 1, Norway). *Scand. J. Gastroenterol.* 12(2):149-159; 1977.

Ig-containing cells were quantitated immunohistochemically in biopsy specimens from the proximal jejunal mucosa of controls and patients with celiac disease (CD). The numbers of IgA, IgM, and IgG immunocytes in a defined "mucosal tissue unit" were, on the average, raised 2.4, 4.6, and 6.5 times, resp., when 13 adult patients with untreated CD were compared with 15 patients who had a

histologically normal mucosa. The IgA:IgM:IgG cell ratios averaged 66:28:6 in untreated CD and 79:18:2.6 in the controls. Similar quantitative data in 10 patients with treated CD were intermediate. IgD- and IgE-containing cells were rare in all patient groups. Most patients in a heterogeneous malabsorption group showed a jejunal Ig-containing cell population similar to that seen in CD, indicating that the local immunocyte pattern may not be specific for the latter disease. The only significant alteration in serum Ig levels related to CD was a raised concentration of IgA compared with normal. This was consistent with the increased amounts of extracellular IgA revealed in the mucosa. However, there was no indication of a defect in the transport of dimeric IgA and IgM through secretory component-producing cells, which in the CD mucosa were present in both crypt and surface epithelium.

1262 IgA CLASS RETICULIN ANTIBODIES IN RELATIVES OF PATIENTS WITH COELIAC DISEASE. (Eng.) Mallas, E. G.; Williamson, N.; Cooper, B. T.; Cooke, W. T. (General Hosp., Birmingham, England). *Gut* 18(8):647-650; 1977.

Patients with various gastrointestinal diseases, patients with celiac disease, and relatives of patients with celiac disease were investigated for IgA class reticulin antibodies, using monospecific anti-IgA and IgG antisera. IgA class reticulin antibodies were not found in any of 19 patients with Crohn's disease, 22 with ulcerative colitis, or 19 with hiatal hernia despite a significant incidence of IgG class reticulin antibodies. None of 56 normal healthy subjects was positive. In contrast, 13 (76%) of the sera from 17 patients with celiac disease on normal diet were positive for IgA class antibodies as were 19 (20%) of 93 first-degree relatives. Seventy-three relatives underwent jejunal biopsy. Grade III (flat) histology was found in 13 and, of these patients, 10 (77%) showed IgA class reticulin antibody in their serum. Determination of IgA class reticulin antibodies is a useful test to determine which relative must be biopsied.

1263 COELIAC DISEASE: THE ABOLITION OF GLIADIN TOXICITY BY ENZYMES FROM Aspergillus niger. (Eng.) Phelan, J. J.; Stevens, F. M.; McNicholl, B.; Fottrell, P. F.; McCarthy, C. F. (Dept. Biochemistry, Univ. Coll., Galway, Ireland). *Clin. Sci. Mol. Med.* 53(1):35-43; 1977.

Xylose absorption, mucosal morphology, and brush border enzymes were studied in patients with celiac disease who ingested carbohydrate-free gliadin, to determine whether the toxicity of gliadin in these patients involves its carbohydrate components. Carbohydrate was removed from dialyzed gliadin by dissolving 50 g in acetic acid, adjusting the pH to 4.5, and adding a mixture of carbohydrases prepared from *Aspergillus niger*. Proteolytic activity was negligible. The gliadin was dialyzed, freeze-dried, and baked into 450-g loaves of bread with gluten-free flour. Three patients in remission each received five loaves of this bread, while a fourth received bread containing untreated gliadin. Only this latter patient showed a definite impairment in xylose absorption. The small intestinal morphology, including villus height, crypt depth, epithelial cell height, and the presence or absence of inflammatory cells in the epithelium and lamina propria, showed no change in the three patients who received enzyme-treated gliadin, while the epithelial cell height and the number of intraepithelial lymphocytes decreased and increased, resp., in the patient who received untreated gliadin. Only this patient showed decreases in all enzymes studied (alkaline phosphatase, lactase, and sucrase). The patient receiving untreated gliadin also had diarrhea, whereas the other three patients did not. The results suggest that carbohydrases present in the microbial extract of *A. niger* are responsible for detoxifying gliadin; however, until a complete characterization of the enzymes involved has been made, the possibility remains that a side-chain substituent other than carbohydrate may account for the toxicity of gliadin.

1264 ACTIVATION OF THE ALTERNATIVE PATHWAY BY GLUTEN: A POSSIBLE AETIOLOGICAL FACTOR IN DERMATITIS HERPETIFORMIS. (Eng.) Massey, A.; Capner, P. M.; Mowbray, J. F. (St. Mary's Hosp. Medical Sch., London, England). *Immunology* 33(3): 339-342; 1977.

Experiments were carried out to determine whether direct activation of the alternative pathway of complement (c) by gluten in the serum is involved in the etiology of dermatitis herpetiformis and celiac disease lesions subsequent to gluten-induced jejunal damage. C3, factor B, and their respective breakdown products were measured by a two-way, antigen-antibody crossed immunoelectrophoresis. In normal serum, there was usually a low level of "spontaneous" C3 conversion. This background level was higher in sera with normal, than in those with reduced, factor B activity. The background was abolished by the presence of EDTA in the incubation mixture. The addition of gluten fractions to normal serum was shown to activate the alternative pathway of complement. C3 and factor B breakdown occurred in a manner analogous to that when activated by zymosan, in the presence of MgEGTA and in serum devoid of classical pathway activity. Bypass activation may be the primary event when gluten enters the serum across a damaged gut mucosa. Immune complexes containing noncomplement-fixing IgA antigluten antibody are carried to the skin, where it is proposed that complexed gluten activates C3 and initiates an inflammatory reaction.

1265 MALABSORPTION SYNDROME, INCLUDING THE CORRELATION WITH THE PANCREATIC DISEASES, WITH PARTICULAR REFERENCE TO THAT WITH ZOLLINGER-ELLISON SYNDROME [Abstract]. (Eng.) Abe, M. (Keio Univ. Sch. Medicine, Tokyo, Japan). *Gastroenterol. Jpn.* 12(4):324-325; 1977.

1266 REFERENCE OF MORPHOLOGICAL CHANGES OF INTESTINAL VILLI AND DISACCHARIDASE ACTIVITY [Abstract]. (Eng.) Iwaki, K. (Nihon Univ. Sch. Medicine, Tokyo, Japan). *Gastroenterol. Jpn.* 12(4):323; 1977.

1267 PATHOPHYSIOLOGY OF MALABSORPTION SYNDROME [Abstract]. (Eng.) Bamba, T.; Hosoda, S. (Kyoto Prefectural Univ. Medicine, Kyoto, Japan). *Gastroenterol. Jpn.* 12(4):321; 1977.

1268 DYSGAMMAGLOBULINAEMIA AND NODULAR LYMPHOID HYPERPLASIA [Abstract]. (Eng.) Barbieri, D.; Koda, Y. K. L.; Yamazake, S. R.; Pasternak, L.; Gama, A. H.; Quarentei, G. (No affiliation given). *Arq. Gastroenterol.* 14(3):158; 1977.

1269 JEJUNAL BIOPSY IN COW'S MILK PROTEIN INTOLERANCE [Letter to Editor]. (Eng.) Walker-Smith, J. A. (St. Bartholomew's Hosp., London EC1, England). *Lancet* 2(8050):1237; 1977.

1270 RIBOFLAVIN DEFICIENCY IN MALABSORPTIVE STATES [Abstract]. (Eng.) Newman, L. J.; Lopez, R.; Cooperman, J. M. (New York Medical Coll., New York, NY 10029). *Fed. Proc.* 37(3):303; 1978.

1271 BACTERIAL OVERGROWTH WITHOUT 'BLIND LOOP' A CAUSE FOR MALNUTRITION IN THE ELDERLY [Abstract]. (Eng.) Roberts, S. H.; Jarvis, E. H.; James, O. (Newcastle General Hosp., Newcastle, England). *Gut* 18(11):A969; 1977.

1272 DUAL ISOTOPE METHOD OF CLINICAL MEASUREMENT OF FAT ABSORPTION USING TRITIATED GLYCEROL TRIETHER--RESULTS IN 101 SUBJECTS [Abstract]. (Eng.) Mackenzie, J. F.; Nelson, L. M.; Robertson, E.; Russel, R. I. (Royal Infirmary, Glasgow, Scotland). *Gut* 18(11):A962; 1977.

1273 OSTEOPOROSIS, IMPAIRED INTESTINAL CALCIUM ABSORPTION, SECONDARY HYPERPARATHYROIDISM, AND INAPPROPRIATELY LOW SERUM 1,25-DIHYDROXYVITAMIN D VALUE--A SYNDROME [Abstract]? (Eng.) Riggs, B. L.; Gallagher, J. C.; DeLuca, H. F.; Edis, A. J.; Lambert, P. W.; Arnaud, C. D. (Mayo Clinic, Rochester, MN). *Clin. Res.* 25(4):623A; 1977.

1274 ABNORMAL SODIUM AND WATER TRANSPORT IN PERNICIOUS ANEMIA--A LUMINAL DEFECT [Abstract]? (Eng.) Arvanitakis, C.; Folscroft, J. (Kansas Univ. Sch. Medicine, Kansas City, KS). *Clin. Res.* 25(4):569A; 1977.

1275 BILIARY SECRETION OF LIPIDS RELATED TO FECAL STEROIDS IN COELIAC DISEASE [Abstract]. (Eng.) Vuoristo, M.; Tarpila, S.; Miettinen, T. A. (Second Dept. Medicine, Univ. Helsinki, Helsinki, Finland). *Scand. J. Gastroenterol.* 12(Suppl. 45):110; 1977.

See also, 0800, 0804, 1030, 1051, 1083, 1084, 1242, 1252, 1259, 1317, 1632, 1646, 1647, 1650.

LARGE INTESTINE

1276 CHANGES IN SITE DISTRIBUTION OF COLORECTAL CARCINOMA IN CONNECTICUT, 1940-1973. (Eng.) Snyder, D. N.; Heston, J. F.; Meigs, J. W.; Flannery, J. T. (Connecticut Cancer Epidemiology Unit, 30 College St., New Haven, CT 06510). *Am. J. Dig. Dis.* 22(9):791-797; 1977.

Because the proportion of colorectal cancer occurring above the rectosigmoid junction has been increasing over time, the 40,771 cases of colorectal carcinoma reported to the Connecticut Tumor Registry from 1940 through 1973 were analyzed to determine the incidence in the ascending, transverse, descending, and sigmoid colon, and the rectum. The number of cases, the proportion of cases, and incidence rates of cancer in the ascending colon and sigmoid have increased over the 34 yr. The percentage of cases of ascending colon carcinoma between 1940 and 1944 was 13.4%, whereas that between 1970 and 1973 was 21.8%. The increases were greatest among people over 65 yr old. Only about half of colorectal cancer can now be identified with the rigid, 25-cm sigmoidoscope. Other tests must be used for early diagnosis of the increased number of cancers in the upper parts of the colon.

1277 MYOTOMY: SURGICAL TREATMENT OF DIVERTICULITIS. (Ger.) Kyrle, P. (Chirurgische Abteilung, Krankenanstalt Rudolfstiftung, Wahringerstr. 27, A 1090 Vienna IX, Austria). *Zentralbl. Chir.* 102(17):1040-1044; 1977.

Sigmoid myotomy was performed in 36 patients with sigmoid diverticulosis (18 cases) and sigmoid diverticulitis (18 cases). About half of the patients had concurrent diseases, such as diabetes mellitus, cardiovascular diseases with hypotension or hypertension, disorders in peripheral arterial circulation, cholecystitis, and sciatica. The original Reilly operation (transection of the tenia libera of the sigmoid colon to the mucosa)

was performed in 34 cases and Hodgson's myotomy (alternating incisions in the tenia libera) in 2 cases. Severe intraoperative and postoperative complications were seen in six cases, including perforation of the mucosa, fistulization, and stenosis of the sigmoid colon. The postoperative mortality was 4/36, due to peritonitis (2), pulmonary embolism (1), and acute pancreatitis (1). The surgical results were good in 29 cases, unsatisfactory in 2, and another patient died of late complications. Exteriorization is recommended in the case of widespread inflammation that makes the separation of the layers of the intestinal wall difficult and increases the risk of mucosal perforation followed by peritonitis. Early carcinoma developing between the diverticula may remain undetected during myotomy, and therefore double-contrast x-ray examination, sigmoidoscopy, and colonoscopy are recommended.

1278 PSEUDOMEMBRANOUS ENTEROCOLITIS IN CHILDHOOD. (Eng.) Buts, J. P.; Weber, A. M.; Roy, C. C.; Morin, C. L. (Hopital Ste-Justine, 3175, Ste-Catherine Rd., Montreal, Quebec H3T 1C5, Canada). *Gastroenterology* 73(14, Part 1):823-827; 1977.

The clinical, laboratory, and histologic features in five children (5-12 yr) with pseudomembranous enterocolitis are presented. The clinical syndrome, characterized by the acute onset of profuse diarrhea, occurred postoperatively in two patients. All patients had received or were taking antibiotics (penicillin, ampicillin, clindamycin). The disease was severe in four children, who presented with hypoproteinemia. Related complications were edema, ascites, pleural effusion, septicemia, and/or shock. The diagnosis was made on the basis of typical rectosigmoidoscopic findings. Despite intensive therapy (i.v. steroids, nafcillin, and gentamicin; plasma infusions; parenteral alimentation), two children died. The use of parenteral nutrition is advocated in severe cases with exudative enteropathy. When profuse diarrhea occurs postoperatively or does not rapidly subside with discontinuation of antibiotic therapy, a high index of suspicion should be maintained, even in children, for the possibility of pseudomembranous enterocolitis. An emergency rectosigmoidoscopy should be done to make an early diagnosis.

1279 DYSTROPHIC EPIDERMOLYSIS BULLOSA. INTERESTING GASTRO-INTESTINAL MANIFESTATIONS. (Eng.) Sehgal, V. N.; Rege, V. L.; Ghosh, S. K.; Kamat, S. M. (Goa Medical Coll., Panaji 403 001, Goa, India). *Br. J. Dermatol.* 96(4):389-392; 1977.

The case of a 20-yr-old woman with a long-standing case of dystrophic epidermolysis bullosa in whom gastrointestinal manifestations occurred is presented. Involvement of the large intestine was characterized by recurrent episodes of diarrhea synchronizing with exacerbation of the skin lesions. The radiological investigations revealed two narrow segments, one each in the descending and transverse colon, with ulcer craters in the lower part of the former. The haustra from the distal half of the transverse colon to the sigmoid colon were lost.

1280 INTERNAL SPHINCTER AND THE NATURE OF HAEMORRHOIDS. (Eng.) Hancock, B. D. (Wythenshawe Hosp., Manchester, England). *Gut* 18(8):651-655; 1977.

To investigate the relationship between an overactive internal anal sphincter and the symptoms of hemorrhoids, internal anal sphincter activity was studied in 84 patients with hemorrhoids and 40 controls without anorectal symptoms. Activity was estimated by measuring maximum resting anal pressure with a water-filled anal balloon probe 7 mm in diameter connected to a strain gauge pressure transducer. There was greater activity of the internal sphincter in patients with hemorrhoids than in controls (107 ± 23.0 versus 85.0 ± 20.5 cm H_2O, $p<0.001$), but there was no significant relation between sphincter activity and duration of symptoms, predominant symptom (bleeding or prolapse), severity of symptoms, history of pain, history of straining at stool, or size of hemorrhoids. Straining at stool occurred significantly more often in patients whose main complaint was prolapse than in those whose main complaint was bleeding ($p<0.025$). Anal dilatation reduced sphincter activity, and the best clinical results were obtained in those with the most active sphincter. An internal sphincter abnormality may be an etiological factor in some patients with hemorrhoids, but there must be other factors as well. Straining at stool may determine whether bleeding or prolapse is the predominant symptom.

1281 METRONIDAZOLE IN THE TREATMENT OF CHRONIC PROCTITIS: A CONTROLLED TRIAL. (Eng.) Davies, P. S.; Rhodes, J.; Heatley, R. V.; Owen, E. (Univ. Hosp. Wales, Cardiff, Wales.) *Gut* 18(8):680-681; 1977.

The effect of metronidazole treatment was tested in a double-blind controlled study of 22 patients with chronic proctitis. In the crossover study, the patients were given metronidazole suppositories (500 mg, 3 times daily) or a placebo in randomized order during two consecutive 28-day periods. The patients recorded the frequency of daily bowel actions and the occurrence of rectal bleeding, and at the end of each period indicated their assessment of their clinical progress. Of the 18 patients completing the trial, 10 noticed no difference between the two periods, 5 improved on placebo, and 3 improved during the metronidazole period. There were no significant differences in stool frequency, blood loss, the sigmoidoscopic appearance of the rectal mucosa, or histologic changes during the two periods. Metronidazole, given for 28 days, does not appear to benefit patients with proctitis, although initial observations in an open trial had been encouraging.

1282 SPHINCTER DENERVATION IN ANORECTAL INCONTINENCE AND RECTAL PROLAPSE. (Eng.)

Parks, A. G.; Swash, M.; Urich, H. (St. Mark's Hosp., London, England). *Gut* 18(8):656-665; 1977.

Biopsies of the external anal sphincter, puborectalis, and levator ani muscles were examined in 6 controls, in 24 women and 1 man with long-standing anorectal incontinence (18 of whom also had rectal prolapse), and in 2 men with rectal prolapse alone. In 16 of the women, anorectal incontinence was of unknown cause, but in 8 there was a history of difficult labor. In all the incontinent patients, there was histological evidence of denervation, which was most prominent in the external anal sphincter muscle biopsies, and least prominent in the levator ani muscles. Myopathic features, which were thought to be secondary, were present in the more abnormal biopsies. There were severe histological abnormalities in small nerves supplying the external anal sphincter muscle in the three cases in which material was available for study. Idiopathic anorectal incontinence may be the result of denervation of the muscles of the anorectal sling, and of the anal sphincter mechanism. This denervation could result from entrapment or stretch injury of the pudendal or perineal nerves occurring as a consequence of rectal descent induced during repeated defecation straining, or from injuries to these nerves associated with childbirth.

1283 CANCER OF THE RECTUM IN OLD AGE, VALUE OF LAPAROSCOPIC INVESTIGATION AND C.E.A. MONITORING [Abstract]. (Eng.) Thorsoe, H. (Silkeborg Centralsygehus, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):106; 1977.

1284 THE PROGNOSIS IN ULCERATIVE PROCTITIS [Abstract]. (Eng.) Sparso, B. H.; Dyring-Andersen, K.; Kragelund, E.; Bonnevie, O. (Hvidovre Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):101; 1977.

1285 A DOUBLE-BLIND STUDY OF METRONIDAZOLE IN PREOPERATIVE TREATMENT BEFORE COLORECTAL SURGERY [Abstract]. (Eng.) Semb, L. S.; Dyrstad, A.; Johnsrud, M. L.; Grabner, P. J.; Viddal, K. O. (Diakonhjemmers Hosp., Oslo 3, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45): 94; 1977.

1286 THE RESPONSE PATTERN OF IMMUNOGLOBULIN-PRODUCING CELLS IN COLON MUCOSA ADJACENT TO CARCINOMA [Abstract]. (Eng.) Rognum, T. O.; Baklien, K.; Brandtzaeg, P. (Rikshospitalet, Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45): 79; 1977.

1287 BLADDER DRAINAGE FOLLOWING SURGERY FOR RECTAL CANCER [Abstract]. (Eng.) Rasmussen, O. V.; Korner, B.; Moller-Sorensen, P.; Kronborg, O. (Bispebjerg Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45): 76; 1977.

1288 IS TRANSVERSOSTOMY PREFERABLE TO COECOSTOMY [Abstract]? (Eng.) Pillgram-Larsen, J.; Haffner, J. F. W.; Normann, E. (Ulleval Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):71; 1977.

1289 DEMONSTRATION OF CANCER CELL SUBPOPULATIONS WITH DIFFERENT DNA CONTENT IN HUMAN COLO-RECTAL CARCINOMAS [Abstract]. (Eng.) Petersen, S. E.; Bichel, P.; Lorentzen, M. (Aarhus Municipal Hosp., Aarhus, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):70; 1977.

1290 RESULTS OF CONTINENT ILEOSTOMY (KOCK'S OPERATION) [Abstract]. (Eng./Ger.) Schaudig, A.; von Bary, S.; Loeschke, K.; Ruckdeschel, G. (Universitatsklinik Munchen, Nussbaumstrasse 20, D-8000 Munchen, W. Germany). *Langenbecks Arch. Chir.* 345:588-589; 1977.

1291 RESULTS OF RECTUM INTRAMURAL LYMPHOGRAPHY [Abstract]. (Eng./Ger.) Junemann, A.; Schacht, U.; Rumpf, P. (Universitatsklinik A, Moorenstr. 4, D-4000 Dusseldorf, W. Germany). *Langenbecks Arch. Chir.* 345:611-612; 1977.

1292 IVALON SPONGE IMPLANTATION IN TREATMENT OF RECTAL PROLAPSE [Abstract]. (Eng./Ger.) Schiller, U.; [illegible]hlke, V. (Klinik fur Allgemeinchirurgie der Univ[illegible]at, Gosslerstrasse 2, D-3400 Gottingen, W. Germany). *Langenbecks Arch. Chir.* 345:612-613; 1977.

1293 THREE YEARS EXPERIENCE WITH THE TRANSSPHINCTERIC MASON APPROACH [Abstract]. (Eng./Ger.) Ruedi, Th.; Durig, M.; Mannhart, H.; Allgower, M. (Kantonsspital, Basel, Switzerland). *Langenbecks Arch. Chir.* 345:612; 1977.

1294 POSTOPERATIVE CARE OF PATIENTS WITH A COLOSTOMY AND THEIR COMPLUCATIONS [Abstract]. (Eng./Ger.) Lange, J.; Geile, D. (Klinikum rechts der Isar der Technischen Universitat, Ismaninger Str. 22, D-8000 Munchen 60, W. Germany). *Langenbecks Arch. Chir.* 345:613; 1977.

1295 TRANSVERSE LOOP COLOSTOMY--A RANDOMIZED TRIAL [Abstract]. (Eng.) Wara, P.; Bone, J. (Arhus Kommunehospital, 8000 Arhus C, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45): 113; 1977.

1296 THE DISTRIBUTION OF POLYPS IN THE LARGE BOWEL: A PROSPECTIVE AUTOPSY STUDY [Abstract]. (Eng.) Vatn, M. H.; Stalsberg, H. (Ulleval Univ. Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):109; 1977.

1297 THE INCIDENCE OF CARCINOMA IN POLYPS REMOVED BY COLONIC POLYPECTOMY [Abstract]. (Eng.) Osnes, M.; Serck-Hanssen, A.; Myren, J.; Qvigstad, T.; Schrumpf, E.; Dybdahl, J.; Melsom, M. (Ulleval Hosp., Oslo 1, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):68; 1977.

1298 IRRITABLE BOWEL SYNDROME ASSOCIATED WITH DIARRHOEA. WATER MOVEMENT, UNIDIRECTIONAL ION FLUXES, AND ELECTRICAL POTENTIAL DIFFERENCE [Abstract]. (Eng.) Oddsson, E.; Rask-Madsen, J.; Krag, E. (Herlev Hosp., Univ. Copenhagen, Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):65; 1977.

1299 CARCINOEMBRYONIC ANTIGEN (CEA) IN THE DIAGNOSIS AND FOLLOW-UP OF COLORECTAL CARCINOMA [Abstract]. (Eng.) Karesen, R.; Hertzberg, J.; Johannesen, J.; Thoresen, B. O.; Orjasaeter, H. (Ulleval Hosp., Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):55; 1977.

1300 SINGLE LAYER COLONIC ANASTOMOSIS [Abstract]. (Eng.) Koudahl, G.; Villumsen, A. (Diakonissestiftelsens Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45): 47; 1977.

1301 CELLULAR AND HUMORAL INDICES OF DISEASE ACTIVITY IN INFLAMMATORY BOWEL DISEASE [Abstract]. (Eng.) Koldkjaer, O.; Klitgaard, N. A.; Schmidt, K. G. (Odense Univ. Hosp., Odense, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45): 46; 1977.

1302 THE RELIABILITY OF THE PATHO-ANATOMICAL DIAGNOSIS OF CANCER OF THE COLON AND RECTUM WITH SPECIAL REFERENCE TO THE CLASSIFICATIONS OF DUKES AND BRODERS [Abstract]. (Eng.) Johansen, A. (Bispebjerg Hosp., Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):42; 1977.

1303 GENERALIZED DERMAL PERIFOLLICULAR FIBROMAS WITH POLYPS OF THE COLON. (Eng.) Hornstein, O. P. (Dept. Dermatology, Univ. Erlangen/Nuremberg, Nuremberg, W. Germany). *Hum. Genet.* 32 (2):193-197; 1976.

1304 PATTERNS OF RECURRENCE FOLLOWING SURGERY ALONE FOR ADENOCARCINOMA OF THE COLON AND RECTUM. (Eng.) Cass, A. W.; Million, R. R.; Pfaff, W. W. (Univ. Florida Coll. Medicine, Gainesville, FL 32610). *Cancer* 37(6):2861-2865; 1976.

1305 TREATMENT OF CHRONIC RADIATION ENTERITIS AND COLITIS WITH SALICYLAZOSULFAPYRIDINE AND SYSTEMIC CORTICOSTEROIDS: A PILOT STUDY. (Eng.) Goldstein, F.; Khoury, J.; Thornton, J. J. (Lankenau Hosp., Philadelphia, PA). *Am. J. Gastroenterol.* 65(3):201-208; 1976.

1306 NEOPLASIA OF THE COLON: A LATE COMPLICATION OF URETEROSIGMOIDOSTOMY. (Eng.) Lindsey, C. M.; Anderson, E. E.; Paulson, D. F. (Duke Univ. Medical Center, Durham, NC). *South. Med. J.* 69(4):501-504; 1976.

1307 POSTOPERATIVE RADIOTHERAPY IN CARCINOMAS OF THE RECTUM AND DISTAL SIGMOID COLON. (Eng.) Mendiondo, O. A.; Wang, C. C.; Welch, J. P.; Donaldson, G. A. (Massachusetts General Hosp., Boston, MA 02114). *Radiology* 119(3):673-676; 1976.

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1309 DIAGNOSIS AND TREATMENT OF EARLY CANCER OF THE RECTUM. (Eng.) Nakagawara, G.; Noguchi, M.; Miyazaki, I. (Sch. Medicine, Kanazawa Univ., Kanazawa, Japan). *Chir. Gastroenterol.* 10(1):63-69; 1976.

1310 PROGNOSTIC SIGNIFICANCE OF PERIPHERAL LYMPHOCYTE COUNTS AND CARCINOEMBRYONIC ANTIGENS IN COLORECTAL CARCINOMA. (Eng.) Kim, U. S.; Papatestas, A. E.; Aufses, A. H., Jr. (Mt. Sinai Sch. Medicine, New York, NY 10029). *J. Surg. Oncol.* 8(3):257-262; 1976.

1311 MANAGEMENT OF FOCALLY MALIGNANT PEDUNCULATED ADENOMATOUS COLORECTAL POLYPS. (Eng.) Shatney, C. H.; Lober, P. H.; Gilbertson, V.; Sosin, H. (Univ. Minnesota Health Science Center, Minneapolis, MN 55455). *Dis. Colon Rectum* 19(4):334-341; 1976.

1312 PLACE OF ELECTROCOAGULATION IN THE TREATMENT OF RECTAL CANCER. (Fre.) Lacour, J.; Prade, M.; Passer, Ph.; Apelbaum, H. (Inst. Gustave Roussy, Villejuif, France). *Bull. Cancer (Paris)* 63(2):191-200; 1976.

1313 A COMBINED THERAPEUTIC APPROACH FOR THE MANAGEMENT OF ADVANCED STAGES OF RECTAL CANCER. (Eng.) Boulis Wassif, S. (Rotterdam Radiotherapy Inst., Rotterdam, Netherlands). *Bull. Cancer (Paris)* 63(2):201-216; 1976.

1314 ALPHA GLOBULINS IN THE SURVEILLANCE OF COLORECTAL CANCER. (Eng.) Cooper, E. H.; Turner, R.; Geekie, A.; *et al.* (Sch. Medicine, Univ. Leeds, Leeds, England). *Biomedicine* 24(3): 171-178; 1976.

1315 POLYPOSIS COLI WITH ADENOCARCINOMA ASSOCIATED WITH CARCINOMA IN SITU OF THE GALLBLADDER. (Eng.) Burney, B.; Assor, D. (Coll. Medicine, Ohio State Univ., Columbus, OH 43210). *Am. J. Surg.* 132(1):100-102; 1976.

1316 PALLIATIVE OPERATIONS FOR RECTAL CANCER. (Rus.) Pokrovsky, G. A.; Brusilovsky, M. I.; Gofman, A. M.; Yulaev, V. N. (Khir. Otd., n.i. Lab. Proktol. Klin., Moscow, USSR). *Khirurgiia (Mosk.)* 52(7):116-120; 1976.

1317 IS THERE SMALL INTESTINAL 'ADAPTATION' AFTER COLECTOMY? A STUDY OF TRANSPORT ACTIVITY IN BIOPSIES OF ILEOSTOMY MUCOSA [Abstract]. (Eng.) Hawker, P. C.; Morris, A. I.; Turnberg, L. A. (Hope Hosp., Manchester, England). *Gut* 18(11):A958; 1977.

1318 BIOPSY DIAGNOSIS OF INFECTIVE COLITIS [Abstract]. (Eng.) Day, D. W.; Mandal, B. K.; Morson, B. C. (St. Mark's Hosp., London, England). *Gut* 18(11):A986; 1977.

1319 CELL KINETIC AND CYCLIC AMP STUDIES IN COLORECTAL CARCINOMA--A GUIDE TO EARLIER DIAGNOSIS [Abstract]? (Eng.) Tregoning, D.; Turner, G.; Watson, A. J.; Britton, D. C.; Bone, G.; Latner, A. L. (Dept. Surgery, Univ. Newcastle upon Tyne, Newcastle upon Tyne, England). *Gut* 18(11):A987; 1977.

1320 RECTAL IgE CELLS IN INFLAMMATORY BOWEL DISEASE [Abstract]. (Eng.) O'Donoghue, D. P.; Kumar, P. J. (St. Bartholomew's Hosp., London, England). *Gut* 18(11):A986; 1977.

1321 IS THERE A MYOELECTRICAL ABNORMALITY IN THE IRRITABLE COLON SYNDROME [Abstract]? (Eng.) Taylor, I.; Darby, C.; Hammond, P.; Basu, P. (Dept. Surgery, Univ. Liverpool, Liverpool, England). *Gut* 18(11):A990; 1977.

1322 SIMILARITY OF BOWEL DISTENSION CHARACTERISTICS IN THE IRRITABLE COLON SYNDROME AND DIVERTICULOSIS [Abstract]. (Eng.) Ritchie, J. (Radcliffe Infirmary, Oxford, England). *Gut* 18(11):A990; 1977.

1323 SPECTRUM OF HEPATIC DYSFUNCTION IN INFLAMMATORY BOWEL DISEASE [Abstract]. (Eng.) Dew, M. J.; Allan, R. N. (General Hosp., Birmingham, England). *Gut* 18(11):A970-A971; 1977.

1324 RADIOTHERAPY IN THE TREATMENT OF CARCINOMA OF THE RECTUM AND COLON. (Fre.) Tawil, E.; Mercier, J. P. (Hopital Maisonneuve, Rosemont, Montreal, Canada). *Union Med. Can.* 105(7):1058-1062; 1976.

1325 SIGMOID VOLVULUS. (Eng.) Moseson, D. L.; Lindell, T.; Brant, B.; Krippaehne, W. (Univ. Oregon Health Science Center, Portland, OR). *Am. Surg.* 42(7):492-497; 1976.

1326 GIANT PSEUDOPOLYPOSIS MIMICKING COLON CANCER [Abstract]. (Eng.) Gruner, O. P. N.; Refsum, S.; Fausa, O.; Hognestad, J. (Rikshospitalet, Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):29; 1977.

1327 PREOPERATIVE BOWEL STERILIZATION WITH NEOMYCIN-BACITRACIN. A CONTROLLED TRIAL [Abstract]. (Eng.) Balslev, I.; Bryde, S.; Andersen, S. B.; Back-Nielsen, P. (Aalborg Sygehus, afsnit Syd, Denmark). *Scand. J. Gastroenterol.* 12(Suppl. 45):6; 1977.

See also, 0781, 0785, 0786, 1040, 1074, 1075, 1081, 1086, 1087, 1088, 1090, 1102, 1111, 1119, 1120, 1159, 1160, 1232, 1235, 1237, 1244, 1255, 1256, 1257, 1259, 1268, 1329, 1331, 1333, 1477, 1628, 1629, 1631, 1633, 1641, 1645, 1651, 1656, 1658, 1661, 1669, 1673, 1679, 1687, 1690.

LARGE INTESTINE

Ulcerative Colitis

1328 EMERGENCY COLECTOMY FOR INFLAMMATORY BOWEL DISEASE. (Eng.) Block, G. E.; Moossa, A. R.; Simonowitz, D.; Hassan, S. Z. (Dept. Surgery, Univ. Chicago, Chicago, IL 60637). *Surgery* 82(4):531-536; 1977.

The results of emergency colectomy for complications of inflammatory bowel disease are described in 53 patients who underwent the operation between 1964 and 1976. All patients were treated consecutively by the authors, on the same surgical service. Thirty-nine patients had ulcerative colitis, 13 Crohn's disease, and 1 pseudomembranous colitis. The indication for operation in 23 patients was hemorrhage; 23 other patients had toxic megacolon, 3 had free perforation, and 4 had fulminant disease. All patients with free perforation or fulminant disease underwent abdominal colectomy; 16 of 23 patients with toxic megacolon received abdominal colectomy, and 7 had proctocolectomy. Seven of 23

patients with hemorrhage underwent proctocolectomy, and 16 underwent abdominal colectomy. The operative mortality rate was 5.6%, and the total mortality rate was 9.4%. The overall mortality rate for the complication of toxic megacolon was 8.7%; following abdominal colectomy, it was 6.1%, and after proctocolectomy, 14.3%. The overall mortality rate for the complication of hemorrhage was 8.7%; following proctocolectomy, it was nil, and after abdominal colectomy, 12.5%. This overall mortality rate compares with the previous decade's mortality rate from the same institutions of 23%. Emergency colectomy is indicated in these patients after prompt and vigorous resuscitative measures and can be performed safely. Proctocolectomy is advocated for those patients whose disease is diagnosed and who suffer massive hemorrhage. Abdominal colectomy is indicated in patients with Crohn's disease with a spared rectum, poor-risk patients with fulminant disease, or patients with toxic megacolon with or without perforation.

1329 ULTRASTRUCTURAL ASPECTS OF COLON EPITHELIUM IN PATIENTS WITH ULCERATED AND GRANULOMATOUS COLITIS. (Ita.) Marinozzi, G.; Familiari, G.; Stillo, F.; Luminari, M.; Giannelli, G. (Istituto di Anatomia Umana Normale dell'Universita di Roma, Rome, Italy). *Minerva Gastroenterol.* 22(4):267-276; 1976.

The epithelium of colonic mucosa was examined by electron microscopy in six patients with ulcerated colitis. Tissue to be examined was obtained by biopsy. Mucosa from patients in the quiescent phase of the disease appeared normal. In patients in the active phase of the disease, the columnar cells were reduced in volume, and interstitial spaces were dilated. Intranuclear inclusion bodies of 400-600 A were seen in all patients. These inclusion bodies may be attributable to herpesvirus. In active cases, round granulomatous bodies of 50-150 A were seen, primarily in the nuclei and occasionally in the nucleoli. Cytoplasmic filaments were also seen in many of the epithelial cells examined. Microfilaments were sometimes noted in the nuclei of cells from patients with active colitis. These filaments are probably the result of mucosal dysplasia.

1330 CLINICAL TRIALS. A LOCAL FOAM AEROSOL IN ULCERATIVE COLITIS. (Eng.) Clark, M. L. (St. Leonard's Hosp., London, England). *Practitioner* 219(1309):103-104; 1977.

The value of 10% hydrocortisone delivered in a foam aerosol in the treatment of distal colitis was examined in 20 patients with ulcerative colitis, Crohn's disease, or nonspecific distal colitis. This treatment was ineffective in three patients; of these, one responded to steroid enemas, while p.o. steroids was necessary in two. Of the 17 patients in whom the foam was effective, 12 had previously received steroid enemas; three of these found the foam enemas more effective than the steroid enemas, while the remainder thought there was no difference between the two treatments. Eleven of the 12 patients who had previously received steroid enemas found that the foam was easier to use. It is concluded that hydrocortisone foam aerosol is a practical and effective form of therapy in cases in which rectal steroids are indicated.

1331 C3 METABOLISM IN ULCERATIVE COLITIS AND CROHN'S DISEASE. (Eng.) Hodgson, H. J. F.; Potter, B. J.; Jewell, D. P. (Massachusetts General Hosp., Boston, MA). *Clin. Exp. Immunol.* 28(3):490-495; 1977.

The metabolism of the third component of complement (C3) was studied in four patients with ulcerative colitis (UC), three patients with Crohn's disease (CD), and seven control subjects. Metabolically functional radioiodinated C3 was isolated from fresh human plasma. The fractional catabolic rates for ^{125}I-labeled C3, expressed as percent of the intravascular pool per hour, were increased ($p<0.0025$) in patients with inflammatory bowel disease (UC, mean 3.01 ± 0.17; CD, mean 3.23 ± 0.14) as compared to the controls (mean 2.05 ± 0.19). The synthesis rate of C3 also increased in UC and CD patients ($p<0.001$). The mean synthesis rate of C3 among CD patients was higher (3.55 ± 0.805 mg/kg/hr) than among UC patients (1.853 ± 0.582 mg/kg/hr, $p<0.05$), and both of these rates were higher than the control rate (1.194 ± 0.110 mg/kg/hr). However, the serum C3 levels were normal (UC, mean 128 ± 20 mg/100 ml) or raised (CD, 184 ± 16 mg/100 ml) in patients with inflammatory bowel disease, the control value being 132 ± 10 mg/100 ml. Thus, there was a significant positive correlation between the fractional catabolic rate of C3 and albumin ($p<0.05$), but no correlation of this rate with serum concentration, among the patients. The results suggest that complement activation may play a role in the pathogenesis of mucosal inflammation in UC and CD.

1332 MENTAL DISTURBANCE IN PATIENTS WITH ULCERATIVE COLITIS [Abstract]. (Eng.) Gruner, O. P. N.; Naas, R.; Gjone, E.; Flatmark, A.; Fretheim, B. (Rikshospitalet, Oslo, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45):28; 1977.

1333 EXTENSION OF INFLAMMATORY CHANGES IN CHRONIC COLITIS ASSESSED BY RADIOLOGY, COLONOSCOPY AND ENDOSCOPIC BIOPSIES [Abstract]. (Eng.) Granqvist, S.; Gabrielsson, N.; Sundelin, P. (Huddinge Univ. Hosp., Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):27; 1977.

1334 ULCERATIVE COLITIS AND PREGNANCY. SURGICAL TREATMENT OF ACUTE COMPLICA-

TIONS. (Ger.) Winkler, R. (Chir. Univ. Klin., Hamburg, W. Germany). *Dtsch. Med. Wochenschr.* 101(25):963-965; 1976.

1335 SURGICAL TREATMENT FOR ULCERATIVE COLITIS. (Jpn.) Tsuchiya. S.; Takemura, H.; Matsuda, Y. (Sch. Medicine, Yokohama City Univ., Yokohama, Japan). *Stomach Intest. (Tokyo)* 11(8): 1005-1014; 1976.

1336 A STATISTICAL SURVEY OF ULCERATIVE COLITIS IN JAPAN. (Jpn.) Yokota, A.; Utsonomiya, T.; Suzuki, K. (Natl. Okura Hosp., Tokyo, Japan). *Stomach Intest. (Tokyo)* 11(8):1023-1028; 1976.

See also, 1262, 1290, 1301, 1318, 1320, 1321, 1323.

PANCREAS

1337 HIGH BREAST-MILK SODIUM IN CYSTIC FIBROSIS [Letter to Editor]. (Eng.) Whitelaw, A.; Butterfield, A. (Queen Charlotte's Maternity Hosp., London W6 OXG, England). *Lancet* 2(8051):1288; 1977.

1338 HETEROZYGOTE DISADVANTAGE IN CYSTIC FIBROSIS [Letter to Editor]. (Eng.) Super, M. (P.O. Box 2619, Windhoek, South West Africa). *Lancet* 2(8051):1288; 1977.

1339 IS PARTIAL DUODENOPANCREATECTOMY JUSTIFIED IN CASES OF CANCER OF THE PANCREAS [Abstract]? (Eng./Ger.) Fuchsjager, N.; Funovics, J.; Fritsch, A. (Univ. Klinik, Alserstrasse 4, A-1090 Wien, Austria). *Langenbecks Arch. Chir.* 345:585; 1977.

1340 VASCULAR PROBLEMS DURING PANCREATECTOMY [Abstract]. (Eng./Ger.) Trede, M.; Kersting, K. H.; Bottger, E. (Klinikum Mannheim der Universitat Heidelberg, Theodor Kutzer-Ufer, D-6800 Mannheim, W. Germany). *Langenbecks Arch. Chir.* 345:585-586; 1977.

1341 WDHA-SYNDROME AND VASOACTIVE INTESTINAL POLYPEPTIDE [Abstract]. (Eng.) Semb, L. S.; Carlsen, E.; Fahrenkrug, J.; Schaffalitzky de Muckadell, O. (Diakonhjemmets Hosp., Oslo 3, Norway). *Scand. J. Gastroenterol.* 12(Suppl. 45): 93; 1977.

1342 THE DIAGNOSIS OF PANCREATIC MALIGNANCIES BY ENDOSCOPIC RETROGRADE PANCREATOGRAPHY (ERCP) COMBINED WITH ENDOSCOPIC RETROGRADE BRUSH CYTOLOGY (ERBC) AND ENDOSCOPIC ASPIRATION CYTOLOGY (EAC) [Abstract]. (Eng.) Osnes, M.; Serck-Hanssen, A.; Swensen, T.; Aune, S.; Myren, J. (Ulleval Hosp., Oslo 1, Norway). *Scand. J. Gastroenterol.* 12 (Suppl. 45):69; 1977.

1343 ALTERED GASTRIC SECRETORY RESPONSES TO A SOLID-LIQUID MEAL IN PANCREATIC INSUFFICIENCY (PI) [Abstract]. (Eng.) Regan, P. T.; Malagelada, J. R.; Go, V. L. W.; DiMagno, E. P. (Mayo Clinic, Rochester, MN). *Clin. Res.* 25(4): 608A; 1977.

1344 DIAGNOSTIC VALUE OF THE ORAL PANCREATIC FUNCTION TEST (PFT) [Abstract]. (Eng.) Mitchell, C. J.; Humphrey, C. S.; Bullen, A. W.; Kelleher, J. (Dept. Medicine and Surgery, Univ. Leeds, Leeds, England). *Gut* 18(11):A979-A980; 1977.

1345 THE LUNDH MEAL AND ERCP IN PANCREATIC DISEASE [Abstract]. (Eng.) Ashton, M. G.; Axon, A. T. R.; Lintott, D. J. (Leeds General Infirmary, Leeds, England). *Gut* 18(11):A980; 1977.

1346 PANCREATIC JUICE ANALYSIS, CARCINOEMBRYONIC ANTIGEN (CEA) AND ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY (ERCP) IN THE DIAGNOSIS OF PANCREATIC DISEASE [Abstract]. (Eng.) Carrlocke, D. L. (Dept. Medicine, Univ. Leicester, Leicester, England). *Gut* 18(11):A980-A981; 1977.

1347 DIFFERENTIATION STAGES OF EXOCRINE CELLS IN THE HUMAN PANCREAS; ULTRASTRUCTURE AND POLYSACCHARIDE CONTENTS [Abstract]. (Eng.) Dutrillaux, M. C.; Hollande, E. (Laboratoire de Physiologie Cellulaire, Universite P. et M. Curie, 4 Place Jussieu, 75005, Paris, France). *Ir. J. Med. Sci.* 146(Suppl. 1):32; 1977.

See also, 0884, 1045, 1090, 1102, 1126, 1127, 1128, 1130, 1231, 1256, 1265, 1545, 1562, 1596, 1632, 1640, 1644, 1674.

1348 ELECTROCARDIOGRAPHIC ALTERATIONS IN ACUTE PANCREATITIS. (Spa.) Bartolome-E., J.; Garnacho, A.; Frison, J. C.; Valle, V.; Rubies-Prat, J. (Ciudad Sanitaria de la Seguridad Social, Universidad Autonoma, Barcelona, Spain). *Rev. Esp. Enferm. Apar. Dig.* 49(4):539-544; 1977.

The electrocardiogram (ECG) patterns of 40 patients treated for acute pancreatitis were studied to evaluate the incidence and establish the type of ECG alterations in these patients. In 18 patients, the ECG's were normal. Abnormal ECG alterations in eight patients could not be properly evaluated. In four patients, ECG irregularities persisted after the acute phase of the disease: in a 70-yr-old man, ECG signs of myocardial necrosis persisted for 2 months with no evidence of elevated enzyme levels; 58-yr-old and 50-yr-old patients with pancreatic disease presented with ECG's characteristic of subepicardial ischemia that persisted for 2 months; and a 9-yr-old patient had an ECG indicative of a first degree auriculoventricular block, which continued for 1 yr after his pancreatitis was cured. Repolarization disorders were found in 10 patients; these disorders returned to normal 2-3 months after after the cure. The recognition of this ECG pattern in acute pancreatitis is an important aid in differentiating between acute pancreatitis and ischemic cardiopathy.

1349 ACUTE PANCREATITIS OF RARE CAUSATION. (Eng.) Pellegrini, C. A.; Acosat, J. M.; Skinner, D. B. (Univ. Chicago Hosp., Chicago, IL). *Surg. Gynecol. Obstet.* 144(6):899-902; 1977.

The cases of eight patients with acute pancreatitis resulting from rare causes are presented. Obstruction of the common pancreatic duct occurred in four patients, and pancreatitis in the remaining four patients resulted from direct parenchymal injury or vascular lesions. In two cases, the ampulla of Vater was obstructed by a foreign body (a T tube in one and a hydatid membrane in the other). In a third patient, obstruction of the pancreatic duct by a tumor resulted in pancreatitis and pseudocyst formation distal to the mass. In a fourth patient, the patency of the pancreatic duct and the more severe and generalized pancreatic injury suggested that mechanisms other than obstruction were involved in the development of acute pancreatitis in association with pancreatic carcinoma. In another patient, pancreatitis was limited to the portion of the pancreas that was to the left of the point of obstruction (a benign stricture of the proximal portion of the main pancreatic duct); a pancreaticojejunostomy provided successful decompression of the obstructed pancreas in this case. In the sixth patient, hyperparathyroidism associated with a large adenoma appeared to be the primary cause of pancreatitis, and this was successfully treated by parathyroidectomy. In the remaining patients, pancreatitis was caused by celiac axis thromboembolism and periarteritis nodosa involving the arterioles of the pancreas, resp. Necrosis occurred in both cases. Surgery is the treatment of choice for pancreatic duct obstruction.

1350 ACUTE PANCREATITIS ASSOCIATED WITH GALLSTONES IN A TWELVE-YEAR-OLD GIRL. (Eng.) Pease, R. (St. James' Hosp., Leeds, England). *Aust. N.Z. J. Surg.* 47(1):87-89; 1977.

The case of a 12-yr-old girl with acute pancreatitis associated with gallstones is presented. The patient had a 1-yr history of intermittent attacks of epigastric pain associated with vomiting. Abdominal examination revealed striking rebound tenderness. Pancreatitis was diagnosed at the first operation, but multiple stones in the gallbladder were not detected, probably because they floated freely within the viscid bile. Three weeks postoperatively, the patient's symptoms returned, and an oral cholecystogram demonstrated multiple small stones. At cholecystectomy, the gallbladder was found to contain 32 small faceted stones. The patient has remained asymptomatic since the second operation. Although pancreatitis is a rare disease in childhood, this diagnosis should be considered in cases of unusual abdominal pain, especially when associated with clinical evidence of ascites.

1351 NUTRITIONAL ASPECTS OF CHRONIC PANCREATITIS IN VERONA, ITALY. (Eng.) Vantini, I.; Cavallini, G.; Angelini, G.; Piubello, W.; Bovo, P.; Benini, L.; *et al.* (III Clinica Medica dell'Universita di Padova, 37100 Verona, Italy). *Rend. Gastroenterol.* 9(1):13-17; 1977.

Nutritional habits before the clinical onset of well-defined chronic relapsing pancreatitis were evaluated in 67 patients from Verona, Italy and 67 paired controls from the same region. The caloric intake of the patients with pancreatitis was significantly higher than that in the controls, both when the total intake (including alcohol) and the alimentary calories were considered ($p<0.001$). The absolute mean intake of proteins, fats, and carbohydrates in the patients (155.9 ± 64.2, 110.5 ± 47.6, and 432.6 ± 192.7 g/day) was significantly higher than that of the controls (112.3 ± 34.6, 77.2 ± 20.8, and 291.6 ± 86.9 g/day, resp., $p<0.001$), but when the data were expressed as percentages of the alimentary calories or of total calories, these differences were not significant.

1352 A SWISS FAMILY WITH HEREDITARY CHRONIC RECURRENT CALCIFYING PANCREATITIS. (Ger.) Bieler, M.; Hammer, B. (Medizinische Klinik, Kantonsspital, CH-9006 St. Gallen, Switzerland). *Schweiz. Med. Wochenschr.* 107(29):1035-1040; 1977.

Hereditary chronic recurrent calcifying pancreatitis in a Swiss family is reported. The disease was confirmed in five patients, the mother and her four children. The age at manifestation of the disease (episodic abdominal pains with emesis) ranged from 8 to 20 yr. Four of the five patients have had diabetes mellitus since the age of 15–40 yr. The fecal fat content was increased in three cases (25.6–53.3 g/day); the fecal chymotrypsin level was 5–48 μg/g versus a normal value of >130 μg/g; and the fecal trypsin level was 2–11 μg/g versus a normal level of >30 μg/g. Lysine and cystine were found in the urine at low, nonpathologic levels. Four other members of the family (the mother's father, her brother, the brother's son, and one of the mother's grandchildren) are suspected of having this disease. Twenty-five families with this disease have been reported in the literature. It is inherited as an autosomal dominant disorder; its pathogenesis is unknown. It is possible that an abnormal pancreatic juice is produced, which causes intracanalicular calcification. The treatment is identical to that of nonhereditary forms, i.e., generally conservative.

1353 EFFECT OF CAERULEIN ON γ-GLUTAMYL TRANSPEPTIDASE AND ALKALINE PHOSPHATASE IN THE DUODENAL JUICE IN CHRONIC PANCREATITIS. (Eng.) Delcourt, A.; Mesa, A.; Wettendorff, P.; Van Melsen, A. (Hopital d'Ixelles, Bruxelles, Belgium). *Digestion* 15(5):385-389; 1977.

The mechanism involved in the abnormally high output of γ-glutamyl transpeptidase (GGT) and alkaline phosphatase (AP) in the duodenal juice in chronic pancreatitis (CP) was investigated in 10 fasted normal controls, 10 patients with chronic pancreatitis, and 3 cholecystectomized patients. The increase of AP and GGT was significantly higher ($p<0.001$) in CP patients than in controls after the administration of cerulein (i.v. for 80 min at 75 ng/kg/hr). This abnormality was not limited to the calcifying type, since among the 10 cases studied only 6 presented pancreatic calcifications. The parallelism of the increase in output of GGT and AP in control and CP patients suggests that same mechanism is involved in both cases. However, the presence of a functioning gallbladder is necessary for this effect to occur, since in cholecystectomized patients and in the patient with T-tube drainage of the biliary tract no increase of GGT was observed after the administration of increasing doses of cerulein in single shots. The effect is not related to the discharge of large amounts of the enzymes by the diseased pancreas. It is suggested that the difference between controls and CP patients is related to an abnormality of the biliary system associated with chronic pancreatitis.

1354 HEREDITARY PANCREATITIS: A FAMILY WITH 4 AFFECTED MEMBERS. (Spa.) Jimenez Caballero, R.; Salinas Carmona, M. C.; Campuzano Fernandez, M.; Perez-Briceno, R.; Lisker Y., R.; Cario, A. (Instituto Nacional de la Nutricion, Av. San Fernando y Viaducto Tlalpan, Mexico 22, D.F.). *Rev. Invest. Clin. (Mex.)* 29(2):165-169; 1977.

1355 RELATIONSHIP BETWEEN HEMOSTASIS AND BLOOD KININS IN PANCREATITIS. (Rus.) Orlov, V. A.; Yakunin, G. A. (Clinical Hosp. No. 20, Moscow, USSR). *Klin. Med. (Mosk.)* 55(8):84-90; 1977.

1356 HEMORRHAGE COMPLICATIONS OF ACUTE PANCREATITIS. (Fre.) Dauchel, J.; Michel, J. M.; Coumaros, D.; Bockel, R.; Marescaux, J.; Sava, G.; Grenier, J. F. (Hospices Civils, 67005 Strasbourg Cedex, France). *J. Chir. (Paris)* 113(2):171-180; 1977.

1357 DIAGNOSIS OF CHRONIC PANCREATITIS. (Ita.) Blum, A. L.; Bruhlmann, W.; Peter, P.; Krejs, G. J. (Hopital Civil Triemli, Zurich, Switzerland). *Minerva Med.* 15(2/3):3621-3634; 1976.

1358 AN EDEMATOUS FORM OF ACUTE PANCREATITIS. (Rus.) Titov, B. M. (12th Municipal Clinical Hosp., Kazan, USSR). *Vestn. Khir.* 117 (7):26-29; 1976.

1359 DUODENAL INTRAMURAL HEMATOMAS OF PANCREATIC ORIGIN. (Fre.) Essioux, H.; Bensaude, R. J.; Cristau, P.; Pailler, J. L.; Molinie, C.; Kassem, A.; *et al.* (Hopital Militaire Begin, F 94160 Saint-Mande, France). *Arch. Fr. Mal. App. Dig.* 65(4):271-275; 1976.

1360 DELAYED OPERATION FOR ACUTE PANCREATITIS. (Ger.) Neher, M.; Kummerle, F.; Mangold, G.; Schonborn, H. (Chirurgische Univ.-Klinik, Langenbeckstrasse 1, D-6500 Mainz 1, W. Germany). *Chirurg* 48(7):439-443; 1977.

1361 SURGICAL TREATMENT OF CHRONIC PANCREATITIS. (Fre.) Sarles, J. C.; Delecourt, P.; Castelo, H. (Hopital D'Aubagne, Avenue du Petit Canedel, Aubagne 13677, France). *Arq. Gastroenterol. Sao Paulo* 14(2):83-88; 1977.

1362 BLOOD SERUM ENZYMES AND ISOENZYMES IN ACUTE PANCREATITIS. (Rus.) Suvalskaya,

L. A.; Bonitenko, Yu. Yu.; Kovalchuk, V. I. (Yu. Yu. Dzhanelidze Scientific Res. Inst. First Aid, Leningrad, USSR). *Klin. Med. (Mosk.)* 55(6):63-67; 1977.

1363 ASPECTS OF PANCREATITIS PATHOGENESIS. (Rus.) Orlov, V. A.; Belyakov, N. V. (Clinical Hosp. No. 20, Moscow, USSR). *Klin. Med. (Mosk.)* 55(6):59-63; 1977.

See also, 0885, 1102, 1126, 1321, 1345, 1346, 1553, 1567, 1674.

LIVER AND BILIARY TRACT

1364 ASSESSMENT OF THE PLASMA DISAPPEARANCE OF CHOLYL-1^{14}C-GLYCINE AS A TEST OF HEPATOCELLULAR DISEASE. (Eng.) Thjodleifsson, B.; Barnes, S.; Chitranukroh, A.; Billing, B. H.; Sherlock, S. (Landspitalinn, Reykjavik, Iceland). *Gut* 18(9):697-702; 1977.

To assess the value of the plasma disappearance of cholyl-1^{14}C-glycine for the detection of hepatic dysfunction, the plasma disappearance of a tracer dose of cholyl-1^{14}C-glycine (10 μCi, 0.2 μmol, i.v.) was examined in 12 control subjects and in 32 patients with hepatocellular dysfunction. Simple analysis of the data did not detect hepatic dysfunction except in the nine patients with severe hepatocellular disease. The greatest degree of discrimination between normal subjects and patients with mild liver disease was obtained by taking the ratio of the plasma retention at 60 min to that at 10 min; it was similar to that obtained with serum γ-glutamyltransferase. The 2-hr postprandial plasma total bile acid concentration gave complete separation between the control subjects and patients with liver disease. This study shows that the plasma disappearance of a tracer dose of cholyl-1^{14}C-glycine is not a sensitive test of hepatic dysfunction.

1365 DIAGNOSTIC USEFULNESS OF PLASMA CARCINOEMBRYONIC ANTIGEN LEVELS IN ACUTE AND CHRONIC LIVER DISEASE. (Eng.) Bullen, A. W.; Losowsky, M. S.; Carter, S.; Patel, S.; Neville, A. M. (St. James' Hosp., Leeds LS9 7TF, England). *Gastroenterology* 73(4):673-678; 1977.

Plasma carcinoembryonic antigen (CEA) levels were measured in 87 patients with liver disease and in 25 controls to assess its value in the diagnosis of acute and chronic liver diseases. Raised plasma CEA levels were found in over 90% of 71 patients with chronic liver disease and 50% of 16 patients with acute liver damage (mean control value=12.1 ± 7.8 ng/ml). Levels increased with increasing chronicity and clinical severity of disease. In individual patients, the plasma CEA level fluctuated with the clinical condition. Persistently normal levels were never found in patients with chronic liver disease in poor clinical condition. A very high level suggests a bad prognosis. If CEA is considered together with the SGPT level, a discriminant function can be calculated, separating patients with acute liver damage and those with an acute exacerbation of chronic active hepatitis, with a high degree of certainty. In acute damage, peak CEA levels occur later than the time of maximum liver necrosis, suggesting that the rise is not owing to release of CEA from damaged cells. There is no significant difference in CEA level between patients with and without spontaneous or surgical portosystemic shunts, suggesting that high levels are not attributable to bypass of the liver. The timing of the rise in CEA in acute liver damage suggests that raised levels may be associated with regeneration. The tendency for higher levels to occur in those patients with greater disturbance of liver function suggests altered metabolism or excretion of CEA.

1366 MODIFICATION OF γ-GLUTAMYL TRANSPEPTIDASE IN LIVER DISEASE. (Ita.) Ragaini, S.; Peracino, A.; Marcovina, S.; Ronchi, E.; Ballini, A.; Mojoli, M. (Ospedale Generale Provinciale di Treviglio e Caravaggio, Italy). *Minerva Med.* 67(43): 2805-2818; 1976.

γ-Glutamyl transpeptidase (GGT) levels were determined in 226 patients, including 20 healthy controls, 118 alcoholics (acute, 18 and chronic, 100), 53 patients with liver cirrhosis, 15 with cholelithiasis without obstructive jaundice, 5 with cholangitis, 6 with chronic aggressive hepatitis (CAH), and 9 with hepatocarcinomas. Increased GGT levels were unusually the result of nonspecific enzyme induction by drugs or alcohol, nonspecific liver damage, or the presence of intra- or extrahepatic cholelithiasis. The majority of patients had one or more of these factors present concurrently. Although elevated GGT levels were present in several conditions, they were determined to be a result of nonspecific induction rather than an indication of the disease state. GGT levels returned to normal when the inducing agent or causative factor was removed from the patient. In CAH and hepatocarcinoma, however, GGT levels were specifically related to the disease state and for a time were the only clinical indications of disease. After correcting for the use of drugs or

alcohol, GGT levels are a precise indication of these two diseases.

1367 CORRELATIONS BETWEEN ANATOMICAL AND CLINICAL FEATURES IN LIVER DISEASE PATIENTS WITH CIRCULATING ANTIBODY OF UNKNOWN ORIGIN. (Ita.) Verme, G.; Rizzetto, M.; Crivelli, O.; Bonino, F. (Ospedale Mauriziano, Turin, Italy). *Minerva Gastroenterol.* 22(3):219-226; 1976.

Antinuclear (AN), anti-smooth muscle (ASM), antimitochondrial (AMT), antimicrosomal (AMC), and antireticular (AR) antibodies were determined by immunofluorescence in 1,000 consecutive patients examined in the gastroenterology division of Maurizian Hospital. Of the 1,000 patients, 267 had liver disease with primary parenchymal involvement; 254 had liver disease with secondary parenchymal involvement, cholelithiasis, or metastases; and 458 had gastrointestinal disease with no evidence of liver involvement. Thirty-three percent of patients with primary parenchymal involvement, 6.3% with secondary parenchymal involvement, and 5.9% with no liver involvement were positive for one or more of the antibodies studied. Of the primary parenchymal involvement patients, 65% had acute hepatitis, 59% had chronic active hepatitis (CAH), 38% had hepatomas, 28% had sclerosing liver disease, and 20% had inactive cirrhosis. ASM antibodies were found in the majority of cases; CAH and inactive cirrhosis patients were AR positive; only CAH patients were AMC positive. ASM antibodies were associated with AN antibodies except in acute hepatitis, where only 4% of the patients were AN positive. In order of frequency, patients were positive for ASM, AN, AMT, AMC, and AR. A subgroup of CAH patients according to a single serological marker could not be identified. There are chronic liver diseases that are distinct from those of viral etiology or metabolic dysfunction and that are most likely autoimmune in origin.

1368 RELATIVE VALUE OF SELECTIVE ARTERIOGRAPHY AND LAPAROSCOPY IN THE DIAGNOSIS OF HEPATIC CARCINOMA. (Spa.) Perez-Jimenez, F.; San Martin, P.; Muro, J.; de Dios, J.; Segura, J. M. (Cuidad Sanitaria de la Seguridad Social "La Paz," Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 47 (1):93-100; 1976.

A comparison is made of selective arteriography and laparoscopy in the diagnosis of hepatocarcinoma. Laparoscopy allows the direct visualization of the abdominal viscera and the detection not only of the existence of a tumor, but also of its size, localization, and nature. On the other hand, since hepatocarcinoma is generally a hypervascularized tumor preferentially nourished by the hepatic artery, arteriography is also an important diagnostic tool. Laparoscopy is contraindicated in cases of cardiopulmonary insufficiency and alterations in the general circulation. On the other hand, arteriography is dangerous when there are alterations in blood coagulation. Moreover, there may be anaphylactic problems, and there is also the inherent risk in the manipulation of arteries with atheromatosis. However, arteriography is generally better tolerated by patients. Arteriography is superior to laparoscopy for the determination of the state of the organ and is a tremendous aid to the surgeon in his planning and handling of a resection; laparoscopy permits only the recognition of the state of the peritoneum and the abdominal viscera accessible to such exploration. The authors conclude that arteriography should be the first diagnostic procedure employed when hepatocarcinoma is suspected. Only when the results of the arteriography are doubtful or when the state of the peritoneum and the resting liver needs to be evaluated for therapeutic purposes should laparoscopy be the final exploratory tool in the systematic diagnosis of hepatic tumors.

1369 CONTRIBUTIONS OF LAPAROSCOPE-DIRECTED BIOPSIES TO THE DIAGNOSIS OF PRIMARY NEOPLASMS OF THE LIVER. (Ita.) Fedeli, G.; Anti, M.; Montano, A. M.; Rapaccini, G. L.; Pizzolato, G. P. (Universita Cattolica del S. Cuore, Istituto di Patologia Speciale Medica e Metodologia Clinica, Rome, Italy). *Minerva Med.* 68(25):1709-1718; 1977.

The use of laparoscopy as a diagnostic tool is discussed. Laparoscope-directed biopsies of the liver are very important in the diagnosis of liver disease, particularly the early diagnosis of neoplasms and in the differentiation of benign and malignant liver tumors. Laparoscopic biopsy reduces diagnosis time and presents little or no risk to the patient. A review of the literature and the authors' personal observations with this technique are presented.

1370 PRIMARY LIVER CELL CARCINOMA (PLCC) IN THE NORTHERN GUINEA SAVANNA OF NIGERIA. (Eng.) Fakunle, Y. M.; Ajdukiewicz, A. B.; Greenwood, B. M.; Edington, G. M. (Dept. Medicine, Ahmadu Bello Univ., Zaria and Kaduna, Nigeria). *Trans. R. Soc. Trop. Med. Hyg.* 71(4):335-337; 1977.

The frequency of primary liver cell carcinoma (PLCC) and the clinical and pathological findings in patients with PLCC were studied in the Guinea Savanna country of Nigeria, an area that has one of the highest frequencies of PLCC in the world. During 1973 and 1974, 185 of 1,170 malignancies were diagnosed as PLCC. The highest frequency occurred among men aged 20-49 yr. Abdominal swelling, pain, hepatomegaly, and ascites were the most common symptoms. Hypoalbuminemia (<3 g%) was present in 67% of the examined cases. Alpha$_1$-fetoprotein occurred in 85% of the 172 patients tested. Hepatitis B surface antigen was detected in 49% of 138 patients. Histologically the tumors were well described and trabecular, with a few showing adenoid or clear cell areas. Liver cell dysplasia was present in almost all cases. These clinical and laboratory findings are similar to those in other series in Africa.

1371 HEMOSTATIC FACTORS IN PRIMARY HEPATOCELLULAR CANCER. (Eng.) van der Walt, J. A.; Gomperts, E. D.; Kew, M. C. (South African Inst. Medical Res., Johannesburg, 2000, South Africa). *Cancer* 40(4):1593-1603; 1977.

Detailed coagulation studies were performed in 19 South African black men with primary hepatocellular cancer (PHC), and the results were compared with those in 19 age-matched controls. Various functional and immunochemical methods were employed in determining the possible presence of functional or structural coagulant protein abnormalities. The patient group was characterized by prolonged prothrombin times ($p<0.001$), partial thromboplastin times ($p<0.001$), and Reptilase times ($p<0.001$); increased levels of fibrinogen, factor VIII, and factor VIII-related antigen; moderately decreased levels of factor V, factor IX, factor X, antithrombin III, and plasminogen; and reduced levels of factor II and factor VII (all $p<0.001$). Functional, immunochemical, and biochemical analysis failed to detect the presence of acquired protein abnormalities. These findings indicate that hemostatic changes in primary hepatocellular cancer are nonspecific in character. Severe alterations in the plasma levels of one or more of these hemostatic factors may occur.

1372 SERUM GLUTAMIC OXALACETIC TRANSAMINASE/GLUTAMIC PYRUVIC TRANSAMINASE RATIOS IN HEPATOCELLULAR CARCINOMA. (Eng.) Shimokawa, Y.; Okuda, K.; Kubo, Y.; Kaneko, A.; Arishima, T.; Nagata, E.; *et al.* (Kurume Univ. Sch. Medicine, Kurume, Japan). *Cancer* 40(1):319-324; 1977.

Changes in SGOT levels and in the SGOT/SGPT ratio were examined in 131 patients with hepatocellular carcinoma (HCC), 76 patients with metastatic liver carcinoma (MLC), and 234 patients with liver cirrhosis. At the onset of the diseases, the SGOT levels in the 39 patients with HCC without cirrhosis, the 92 patients with HCC with cirrhosis, the 234 with cirrhosis alone, and the 76 with metastatic carcinoma were 96 ± 12, 152 ± 14, 84 ± 17, and 64 ± 17 U, resp. The SGPT levels were 42 ± 5, 54 ± 5, 55 ± 4, and 38 ± 5 U, resp., and the SGOT/SGPT ratios were 2.5 ± 0.2, 3.5 ± 0.3, 1.9 ± 0.1, and 2.2 ± 0.1, resp. The differences in SGOT and SGOT/SGPT ratio between the patients with cirrhosis and those with HCC and cirrhosis were highly significant ($p<0.01$). In all patients with HCC, there was a continuous increase in the SGOT level 2-3 months before death, while SGPT remained almost unchanged. In the patients with MLC, a similar, but less prominent change occurred in the preterminal period. Although the LDH activities increased in all groups during the 2 months before death, this was most striking in MLC. The average SGPT level was lower in HCC patients without cirrhosis at autopsy, compared with those with HCC and cirrhosis ($p<0.01$), making the SGOT/SGPT ratio greater in the former patients. The SGPT level was lower in patients with a liver weighing more than 3,500 g than that in patients with a liver weighing less than 1,500 g; since the SGOT level was not much different, the SGOT/SGPT ratio was greater in the former ($0.05> p>0.1$).

1373 MULTIPLE TUMORS AFTER ANDROGEN THERAPY. (Eng.) Sale, G. E.; Lerner, K. G. (Fred Hutchinson Cancer Res. Center, 1124 Columbia St., Seattle, WA 98104). *Arch. Pathol. Lab. Med.* 101 (11):600-603; 1977.

A case of multiple hepatomas with early peliosis hepatis, multiple pancreatic islet cell tumors, and a renal medullary interstitial cell tumor following 5 yr of androgen and prednisone therapy for idiopathic aplastic anemia is reported. The patient, a 37-yr-old man, died shortly after allogeneic bone marrow transplantation. The hepatic tumors were small and well differentiated, and the pancreatic tumors were of mixed ribbon and islet cell pattern. During the 5-yr period, the patient had received testosterone, oxymetholone, methyltestosterone, fluxymesterone, and prednisone. The tumors in this case probably represent part of a continuum similar to those induced in experimental rats by a combination of carcinogen and androgens, and it would thus seem prudent to designate the hepatomas as adenomas or as hepatomas with variable malignant potential. Exploration of a possible trophic role of androgens or corticosteroids for the renomedullary interstitial cell may be warranted for the study of the renal tumor in this patient.

1374 HEPATOPORTAL ARTERIOVENOUS FISTULA IN PRIMARY CARCINOMA OF THE LIVER. (Eng.) Nagasue, N.; Inokuchi, K.; Kobayashi, M.; Saku, M. (Kyushu Univ. Sch. Medicine, Fukuoka, Japan). *Surg. Gynecol. Obstet.* 145(4):504-508; 1977.

The correlation between portal circulation and esophageal varices was evaluated in 80 patients with a primary hepatic carcinoma, with special regard to the significance of the presence of hepatoportal arteriovenous fistula. Hepatoportal arteriovenous fistula was found in eight patients. Esophageal varices were observed in 32 of the patients with a hepatoma. Seventeen of 52 patients without hepatoportal arteriovenous fistula had varices compared with all patients with a fistula. A fatal hemorrhage was encountered in five patients with the fistula. A rational treatment for those with the triple lesions (hepatoma, esophageal varices, and hepatoportal arteriovenous fistula) may be a ligation of the hepatic artery.

1375 BENIGN HEPATOCELLULAR TUMORS. (Eng.) Guzman, I. J.; Gold, J. H.; Rosai, J.; Schneider, P. D.; Varco, R. L.; Buchwald, H. (Univ. Minnesota Medical Sch., Minneapolis, MN 55455). *Surgery* 82(4):495-503; 1977.

All cases diagnosed as benign hepatocellular tumors occurring at the University of Minnesota Hospitals from 1950 to 1976 are summarized, and the tumor pathology, clinical aspects, and possible association with the use of oral contraceptives are discussed. Excluding hemangiomas, 40 tumors were reviewed and reclassified. Eighteen were diagnosed as focal nodular hyperplasia (FNH), 10 as hepatocellular adenomas (HCA), 11 as bile duct adenomas (BDA), and 1 as a mesenchymal hematoma. Among the patients

with FNH, the female:male ratio was 8:1, and the mean age was 43 ± 18 yr. Two patients were symptomatic; of these, one presented with portal hypertension. Treatment consisted of local resection in 12, left hepatectomy in 1, and right hepatectomy in 2. Eight patients had a history of oral contraceptive or other steroid intake. The tumors occurring after 1964 were larger ($p<0.05$). In the patients with HCA, the female:male ratio was 4:1, and the mean age was 33 ± 20 yr. Five tumors were found at necropsy; the other five were symptomatic. Treatment consisted of local resection in one patient, right hepatectomy in two, and left hepatectomy in one. Three patients had a positive history of oral contraceptive intake. There was no statistically significant difference in the size or the incidence of hepatocellular adenomas between 1950 to 1963 and 1964 to 1976. In BDA, the female:male ratio was 7:4, and the mean age was 57 ± 15 yr. Only one case was symptomatic, and the lesion in this patient measured 10 cm. The other BDA's were 1 cm or less in diameter. There is no suggestion that these tumors are associated with use of birth control pills. This series has a lower proportion of symptomatic patients and of patients on oral contraceptives than is suggested by reviewing the literature. It is concluded that a causal relationship between use of oral contraceptives, or other steroids, and benign hepatocellular tumors cannot be demonstrated. However, this conclusion does not rule out that oral contraceptives, or other steroids, might have a contributory influence on the initiation of certain of these tumors or on tumor size.

1376 SERUM-FERRITIN IN DIAGNOSIS OF HAEMOCHROMATOSIS: A STUDY OF 43 FAMILIES. (Eng.) Halliday, J. W.; Cowlishaw, J. L.; Russo, A. M.; Powell, L. W. (Royal Brisbane Hosp., Brisbane, 4029, Queensland, Australia). *Lancet* 2(8039): 621-623; 1977.

Two hundred forty-two members of 43 families with idiopathic hemochromatosis were investigated for increased body-iron stores to assess the value of serum ferritin determination as a screening test to detect preclinical disease. The serum iron concentration was elevated in only 76% of relatives with increased iron stores, and it was also elevated in 10% of relatives with normal iron stores. The percentage saturation of transferrin was elevated in all relatives with increased iron stores but also in 33% of relatives with normal iron stores. Serum ferritin was raised in 98% of relatives with increased iron stores and in only three (1.8%) of those with normal iron stores. These three subjects consumed alcohol in excess of 100 g ethanol daily, and their serum ferritin levels fluctuated widely. Increased iron stores were reflected in increased serum ferritin concentrations in subjects as young as 14 yr in whom the liver iron concentration was twice the normal upper limit and before there was any evidence of architectural damage to the liver. The serum ferritin concentration is a useful noninvasive screening test for precirrhotic hemochromatosis.

1377 INFLUENCE OF DEOXYCHOLIC ACID ON BILIARY LIPIDS IN MAN. (Eng.) Ahlberg, J.; Angelin, B.; Einarsson, K.; Hellstrom, K.; Leijd, B. (Karolinska Institutet at Serafimerlasarettet, Stockholm, Sweden). *Clin. Sci. Mol. Med.* 53(3): 249-256; 1977.

To gain information about the possible influence of deoxycholic acid on the composition of bile, the duodenal bile acid composition was analyzed in 24 control subjects and 107 patients with various types of hyperlipoproteinemia. A highly significant negative correlation was observed between the proportions of deoxycholic acid and cholic acid ($r=-0.80$, $p<0.001$) as well as between deoxycholic acid and chenodeoxycholic acid ($r=-0.58$, $p<0.001$). The patients with gallbladder disease had an increased proportion of deoxycholic acid in their bile. Eight controls were studied before and during the ingestion of 1.9 mmol (0.75 g) of deoxycholic acid daily. In these subjects, a rise in the proportion of deoxycholic acid was also accompanied by a fall in the proportion of both cholic acid and chenodeoxycholic acid in duodenal bile. The biliary lipid composition and cholesterol saturation was determined before and during the administration of 1.9 mmol (0.75 g) of chenodeoxycholic acid (12 patients) or deoxycholic acid (8 patients) daily for 3-4 weeks. The cholesterol saturation was decreased during the chenodeoxycholic acid ingestion (from 10.2 ± 0.7% of total bile lipids to 7.2 ± 0.7%, $p<0.001$), whereas no change occurred in bile saturation during deoxycholic acid administration. The ingestion of chenodeoxycholic acid lowered serum triglyceride, and deoxycholic acid lowered the serum cholesterol. The mechanisms and significances of these findings remain to be determined.

1378 THE IMMUNOELECTRON-MICROSCOPICAL DEMONSTRATION OF ANTIBODIES AGAINST ENDOPLASMIC RETICULUM (MICROSOMES) IN CHRONIC AGGRESSIVE HEPATITIS AND LIVER CIRRHOSIS. (Eng.) Storch, W.; Cossel, L.; Dargel, R. (Neurologischen Klinik der Karl-Marx-Universitat, DDR-7039, Leipzig, Karl-Marx-Stadter Str. 50, A 2, E. Germany). *Immunology* 32(6):941-945; 1977.

Indirect immunoelectron microscopic observations made on microsomal fractions of rat liver treated with microsomal antibodies obtained from the blood of patients with liver cirrhosis and hepatitis and incubated with peroxidase-labeled antihuman IgG are reported. Microsomal antibodies were detected in the sera of 18 subjects among 1,911 patients included in the investigation. Seven of these 18 patients had chronic aggressive hepatitis, 1 had persistent hepatitis, and 10 had liver cirrhosis. In microsomal fractions treated with serum antibodies from these patients, the fragments of endoplasmic reticulum (microsomes) were less densely packed and less contrasting than in control microsomal fractions that were not treated with sera from patients with microsomal antibodies. Extremely fine granular to amorphous (homogenous) material of high electron density was observed to be localized at the sites of microsomal membranes

(fragments of endoplasmic reticulum) so that ring-shaped structures (or vesicles) having relatively thick-walled borders of different width and high electron density were produced. These results were not obtained in parallel tests with control microsomal fractions that were not treated with the microsomal antibodies. The fine granular to amorphous deposits of high electron density may be the substrate of an antibody reaction against membranes of the endoplasmic reticulum, made visible by the aid of peroxidase-labeled antibodies. The results suggest that the antibody reaction is chiefly directed towards the membranes of the endoplasmic reticulum.

1379 NONFUNCTIONING ARGYROPHILIC TUMOR (APUDoma) OF THE HEPATIC DUCT. SIMPLIFIED METHODS OF DETECTING BIOGENIC AMINES IN TISSUE. (Eng.) Judge, D. M.; Dickman, P. S.; Trapukdi, S. (Milton S. Hershey Medical Center, Hershey, PA 17033). *Am. J. Clin. Pathol.* 66(1):40-45; 1976.

1380 LIVER DISEASE IN HEMOPHILIA [Abstract]. (Eng.) Van Thiel, D. H.; Spero, J. A.; Rabin, B. S.; Lewis, J. H. (Univ. Pittsburgh Sch. Medicine, Pittsburgh, PA). *Gastroenterology* 73(5): 1252; 1977.

1381 THE OCCURRENCE OF CEREBRAL AIR EMBOLISM AND OTHER NEUROLOGICAL COMPLICATIONS AFTER LIVER TRANSPLANTATION [Abstract]. (Eng.) Starzl, T. E.; Schneck, S. A.; Mazzoni, G.; Aldrete, J. A.; Porter, K. A.; Schroter, G. P. J.; *et al.* (Univ. Colorado Medical Center, Denver, CO). *Gastroenterology* 73(5):1248; 1977.

1382 CARCINOEMBRYONIC ANTIGEN AND PHOSPHOHEXOSE ISOMERASE, GAMMAGLUTAMYL TRANSPEPTIDASE AND LACTATE DEHYDROGENASE LEVELS IN PATIENTS WITH AND WITHOUT LIVER METASTASES. (Eng.) Munjal, D.; Chawla, P. L.; Lokich, J. J.; Zamcheck, N. (Boston City Hosp., Boston, MA 02118). *Cancer* 37(4):1800-1807; 1976.

1383 METABOLIC INHIBITION OF POLYMORPHONUCLEAR LEUCOCYTES IN FULMINANT HEPATIC FAILURE. (Eng.) Bailey, R. J.; Woolf, I. L.; Cullens, H.; Williams, R. (King's Coll. Hosp. Medical Sch., London, England). *Lancet* 1(7970):1162-1163; 1976.

1384 PLASMA CYCLIC ADENOSINE 3',5' MONOPHOSPHATE RESPONSE TO GLUCAGON IN PATIENTS WITH LIVER DISEASE. (Eng.) Davies, T. F.; Prudhoe, K.; Douglas, A. P. (Royal Victoria Infirmary, Newcastle upon Tyne, England). *Br. Med. J.* 1(6015):931-933; 1976.

1385 HEPATO-NEPHROMEGALIA GLYCOGENICA (GLYCOGEN STORAGE DISEASE OF THE LIVER AND KIDNEYS). (Ger.) Von Gierke, E. (Stadt. Krankenh. Karlsruhe i.B., W. Germany). *Beitr. Pathol.* 158(1): 94-108; 1976.

1386 LIVER FUNCTION AND INH AND PAS METABOLISM IN ADOLESCENTS WITH SIDE-EFFECTS FROM CHEMOTHERAPY FOR TUBERCULOSIS. (Rus.) Tytor, Ya. N.; Koropetsky, V. I. (Ternopol Regional Medical Inst., Ternopol, USSR). *Vrach. Delo* (6):61-64; 1977.

1387 CONSERVATIVE TREATMENT OF CAVO-HEPATIC TRAUMATIC DISINSERTION: REPORT OF ONE CASE. (Fre.) Migliori, G.; Michelangeli, F.; Pras, N.; Fombaron, G.; Inglesakis, J. A. (Hopital Saint-Roch, 5, rue Pierre-Devoluy, 06000 Nice, France). *J. Chir. (Paris)* 113(5/6):487-496; 1977.

1388 SURGICAL MANAGEMENT OF LIVER TRAUMA. (Ger.) Klosoris, E.; Siedek, M.; Kuhr, J.; Kozuschek, W. (Chirurgische Universitatsklinik und Poliklinik, D-5300 Bonn-Venusberg, W. Germany). *Langenbecks Arch. Chir.* 344(1):61-70; 1977.

1389 MINIREVIEW: ENDOTOXIN EFFECTS ON THE LIVER. (Eng.) Utili, R.; Abernathy, C. O.; Zimmerman, H. J. (Veterans Admin. Hosp., Washington, DC 20422). *Life Sci.* 20(4):553-568; 1977.

1390 DIAGNOSIS AND SURGICAL TREATMENT FOR MULTILOCULAR ECHINOCOCCOSIS OF THE LIVER. (Eng.) Kasai, Y.; Sasaki, E.; Tamaki, A.; Koshino, I.; Kakita, A.; Kawanishi, N. (Hokkaido Univ., Sch. Medicine, Sapporo, Japan). *Chir. Gastroenterol.* 10(4):425-428; 1976.

1391 A STUDY OF BILE VISCOSITY IN HEPATO-BILIARY DISEASES AND INFLUENCE OF ALTERATION OF BILE VISCOSITY IN VIVO. (Eng.) Nakamoto, M.; Nakayoshi, A.; Sato, Y.; Takahashi, T.; Ide, T.; Nagao, F. (Jikei Univ. Sch. Medicine, Japan). *Chir. Gastroenterol.* 10(4):445-451; 1976.

1392 MULTIFOCAL MALIGNANT HEMANGIOENDOTHELIOMA PRESENTING AS ACUTE HEPATITIS: A CLINICOPATHOLOGIC STUDY. (Eng.) Stein, A. M.; Fawaz, K.; Tabrizi, A.; Blanchard, R.; Hou, S. (New England Medical Center Hosp., 171 Harrison Ave., Boston, MA 02111). *Am. J. Gastroenterol.* 67(4):370-374; 1977.

1393 SUCCESSFUL TREATMENT OF EARLY CANCER OF THE LIVER AND PORTAL HYPERTENSION IN PATIENTS PRESENTING WITH BLEEDING OESOPHAGEAL VARICES. (Eng.) Kobayashi, M.; Inokuchi, K.; Nagasue, N.; Saku, M.; Iwaki, A. (Kyushu Univ. Hosp., Fukuoka 812, Japan). *Br. J. Surg.* 64(8):542-544; 1977.

1394 ANGIOGRAPHIC DEMONSTRATION OF GROWTH OF HEPATOCELLULAR CARCINOMA IN THE HEPATIC

VEIN AND INFERIOR VENA CAVA. (Eng.) Okuda, K.; Jinnouchi, S.; Nagasaki, Y.; Kuwahara, S.; Kaneko, T.; Kubo, Y.; *et al.* (Chiba Univ. Sch. Medicine, Chiba 280, Japan). *Radiology* 124(1):33-36; 1977.

1395 RADIOISOTOPE SCANNING IN EVALUATION OF METASTATIC DISEASE IN THE LIVER. (Eng.) Rozin, R.; Pavlovski, B.; Merhav, A. (Hadassa Hosp., Tel Aviv, Israel). *Abdom. Surg.* 19(7/8):178-180; 1977.

1396 CARCINOMA ARISING IN A LIVER CYST--REPORT OF THREE CASES. (Eng.) Kasai, Y.; Sasaki, E.; Tamaki, A.; Koshino, I.; Kawanishi, N.; Hata, Y. (Hokkaido Univ. Sch. Medicine, Sapporo, Japan). *Jpn. J. Surg.* 7(2):65-72; 1977.

1397 NEW ASPECTS OF CLINICAL HEPATOLOGY. (Ger.) Gerok, W. (Med. Klinik der Universitat, Hugstetter Str. 55, D-7800 Freiburg, W. Germany). *Munch. Med. Wochenschr.* 119(16):529-534; 1977.

1398 LIVER METABOLISM OF DRUGS. (Ita.) Darnis, F. (Hopital Beaujon, 100, Bd. du General Leclerc, 92-110 Clichy, France). *Minerva Med.* 67 (55):3591-3598; 1976.

1399 DIAGNOSTIC ACCURACY OF TRANSUMBILICAL PORTOGRAPHY. (Cze.) Cholt, M.; Kalny, J.; Spatenka, J. (Rentgenologicke pracoviste katedry fakultni pediatrie fakulty detskeho lekarstvi KU, Prague, Czechoslovakia). *Cesk. Radiol.* 31(2):91-98; 1977.

1400 THE USE OF PATHOPHYSIOLOGICAL SYNDROME CHARACTERISITICS FOR EVALUATING THE FUNCTIONAL STATE OF THE LIVER IN CHILDREN. (Rus.) Volkova, A. A. (Gorki Scientific Res. Inst. Pediatrics, Gorki, USSR). *Pediatriia* (3):54-56; 1977.

1401 USE OF ^{131}I-ALBUMIN MICROAGGREGATES FOR THE ASSESSMENT OF PROTEOLYTIC AND PHAGOCYTIC FUNCTIONS OF THE LIVER. (Rus.) Gabuniya, R. I.; Ikonnikov, A. I. (Scientific Res. Inst. Medical Radiology of USSR Acad. Medical Sciences, Obninsk, USSR). *Med. Radiol. (Mosk.)* 22(5):3-7; 1977.

1402 SIGNIFICANCE OF RHEOGRAPHY IN DIAGNOSIS OF CHRONIC NONPROGRESSIVE AND PROGRESSIVE DISEASES OF THE LIVER. (Rus.) Serdyuk, N. N. (Univ. Clinic, Ivano-Frankovsk Medical Inst., Ivano-Frankovsk, USSR). *Ter. Arkh.* 49(5):110-113; 1977.

1403 MANIFESTATIONS OF AUTOIMMUNITY IN CHRONIC LIVER DISEASES. (Cze.) Horejsi, J.; Jezkova, Z.; Svorcova, V.; Novak, J.; Laznicka, M. (Ustav Hematologie a Krevni Transfuze, Prague, Czechoslovakia). *Cas. Lek. Cesk.* 115(37):1146-1149; 1976.

See also, 0895, 0916, 0920, 0958, 0968, 0988, 1089, 1090, 1091, 1092, 1093, 1100, 1104, 1106, 1112, 1113, 1114, 1132, 1133, 1135, 1136, 1137, 1138, 1140, 1142, 1145, 1222, 1231, 1234, 1260, 1271, 1323, 1404, 1422, 1425, 1451, 1458, 1474, 1475, 1478, 1487, 1488, 1506, 1526, 1532, 1537, 1541, 1584, 1623, 1662, 1674, 1676, 1683.

LIVER AND BILIARY TRACT

Hyperbilirubinemic States

1404 LIVER DISEASE IN INFANTS. PART II: HEPATIC DISEASE STATES. (Eng.) Mathis, R. K.; Andres, J. M.; Walker, W. A. (Massachusetts Hosp., Boston, MA 02114). *J. Pediatr.* 90(6):864-880; 1977.

A review is presented of the developmental and acquired disorders of bilirubin metabolism (other than hepatic excretory defects) in infants. Noncholestatic jaundice (unconjugated hyperbilirubinemia) of various types (e.g., physiologic jaundice), jaundice due to overproduction of bilirubin, and jaundice due to impaired transport, hepatic uptake or conjugation, or enterohepatic circulation of bilirubin are discussed together with the treatment of these entities. Different types of cholestatic jaundice (conjugated hyperbilirubinemia) are described: idiopathic cholestatic jaundice, inherited cholestatic jaundice (familial hepatocellular cholestasis and metabolic hepatocellular cholestasis), inherited "noncholestatic" jaundice, and acquired cholestatic jaundice. Infectious liver disease of infants (e.g., viral hepatitis, sepsis, and abscess) and iatrogenic liver disease due to chemical liver injury, i.v. alimentation, or injury due to catheter placement in umbilical vessels, or ionizing irradiation are also described. It is suggested that every step in the metabolism of bilirubin can be affected by a spectrum of conditions causing jaundice and that hyperbilirubinemia in neonates and infants

should be regarded as abnormal until proven otherwise.

1405 UNCONJUGATED BILIRUBIN AND AN INCREASED PROPORTION OF BILIRUBIN MONOCONJUGATES IN THE BILE OF PATIENTS WITH GILBERT'S SYNDROME AND CRIGLER-NAJJAR DISEASE. (Eng.) Fevery, J.; Blanckaert, N.; Heirwegh, K. P. M.; Preaux, A. M.; Berthelot, P. (Dept. Medical Res., Univ. Leuven, 300 Leuven, Belgium). *J. Clin. Invest.* 60(5):970-979; 1977.

Bilirubin pigments were studied in the bile of 20 normal adults, 25 patients with Gilbert's syndrome, 9 children with Crigler-Najjar disease, and 6 patients with hemolytic diseases (congenital spherocytosis or autoimmune hemolysis), to determine how hepatic bilirubin uridine diphosphate (UDP)-glucuronosyltransferase deficiency affects the end products of bilirubin biotransformation. In bile from patients with Gilbert's syndrome, there was a striking increase in the proportion of bilirubin monoconjugates (48.6 ± 9.8% of total conjugates) relative to that in normal bile (27.2 ± 7.8%). This increase was even more pronounced in children with Crigler-Najjar disease, in whom bile glucuronide was always demonstrated. Furthermore, unconjugated bilirubin-IXα was always present in the bile of these children and amounted to 30-57% of their total bilirubin pigments (<1% in the controls). It was not possible to predict from the biliary bilirubin composition whether a child would respond to phenobarbital therapy. Bile composition was normal in patients with hemolysis, except when there was associated deficiency of hepatic glucuronosyltransferase. Therefore, the observed alterations were not a simple consequence of unconjugated hyperbilirubinemia. The findings suggest that Crigler-Najjar disease represents a more pronounced expression than Gilbert's syndrome of a common biochemical defect. Hepatic bilirubin UDP-glucuronosyltransferase deficiency leads to decreased formation of diconjugates with an ensuing increase in the proportion of bilirubin monoconjugates in bile; in the most severe cases, an elevated content of biliary unconjugated bilirubin is also found.

1406 PHYSIOPATHOLOGICAL AND THERAPEUTIC FEATURES OF GILBERT'S ICTERUS AT THE PRESENT TIME. I. RECENT FINDINGS IN GILBERT'S SYNDROMES. (Ita.) Lovisetto, P.; Giorcelli, W.; Actis, G. C.; Biarese, V. (Istituto di Clinica Medica Generale e Terapia Medica I, Universita degli Studi di Torino, Turin, Italy). *Minerva Med.* 67(35):2253-2265; 1976.

Gilbert's icterus is a term used to describe some forms of free-bilirubin hyperbilirubinemia that are caused by a defect in bilirubin clearance by liver cells. Gilbert's icterus occurs in 4-6% of the population and appears to be an inherited metabolic disturbance in which there is a reduction in glucuronyl transferase activity. In certain individuals, there is an alteration in the capacity of the liver cells to retain bilirubin that has been removed from the blood. Gilbert's icterus is of two types: one with slight bilirubinemia that is a result of defective uptake and one with higher bilirubin that is due to a reduction in glycuronide conjugation. Lysosome-like granules are seen at the biliary pole of the liver in Gilbert's syndrome patients. The role of these granules in the disease process is not known. Clinical examination sometimes reveals slight hepatomegaly and a more or less intense icterus. On laboratory examination, the average life of erythrocytes may be shortened and kinetic studies with free bilirubin or with radiolabeled bilirubin may show alterations in a selected parameter. Most Gilbert's icterus patients are asymptomatic, however.

1407 PHENOBARBITAL PROPHYLAXIS OF NEONATAL HYPERBILIRUBINEMIA: EVALUATION OF A NIGHTLY DOSE. (Eng.) Thomas, C. R. (Southside OB/GYN, Inc., 3530 South Keystone Ave., Suite 111, Indianapolis, IN 46227). *Obstet. Gynecol.* 50(5): 607-610; 1977.

An attempt was made to improve the success of a previously described method of preventing neonatal hyperbilirubinemia. Two hundred twelve women received phenobarbital in a single 30-mg nightly dose from 32 weeks of gestation until delivery, and the incidence of jaundice in their infants was compared with that in the infants of 254 untreated mothers. A 39% reduction in significant hyperbilirubinemia was realized, but this fell far short of previous findings with a larger dose. Premature infants received no apparent protection against jaundice. No mature infants had severe jaundice (>16 mg/100 ml), and the resolution of hyperbilirubinemia in this group was more rapid. No apparent difference in drowsiness or daytime sedation from previous experience was found. Female newborns responded much better (3:1) to prophylaxis. These disappointing results favor a return to the 1-g daily divided dose.

1408 IMPAIRED DRUG CONJUGATION IN SUBJECTS WITH GILBERT'S SYNDROME (GS) [Abstract]. (Eng.) Kutz, K.; Schulte, A.; Jensen, C.; Gugler, R. (Medizinische Poliklinik, Univ. Bonn, Bonn, W. Germany). *Gastroenterology* 73(5):1229; 1977.

1409 GILBERT'S SYNDROME: THE QUALITATIVE BASIS OF THE DISORDER [Abstract]. (Eng.) Gordon, E. R.; Shaffer, E. A.; Pare, P.; Goresky, C. A. (Montreal General Hosp., Montreal, Quebec H3G 1A4, Canada). *Gastroenterology* 73(5):1222; 1977.

See also, 0963, 0964, 1078.

Nonviral Hepatitis and Hepatic Injury

1410 HEPATOTOXICITY OF ANESTHETICS. (Spa.) Hernandez, A. M. (Hospital Provincial, Murcia, Spain). *Rev. Esp. Enferm. Apar. Dig.* 47 (6):785-794; 1976.

The metabolism of anesthetics by the liver and the resultant hepatotoxicity of these agents are reviewed. The anatomy and normal physiological functions of the liver are described with special emphasis on drug metabolism. It is concluded that the majority of anesthetics may be considered "protoplasmic poisons." The types of hepatic lesions commonly associated with hepatic diseases and the diagnostic tests for hepatic function are enumerated. The choice of the type of anesthetic to use, particularly for a patient with a disease of the liver, requires careful consideration of the drugs that the liver will metabolize to excrete the anesthetic, and the use of anesthetics that are potentially hepatotoxic (chloroform, halothane, methoxyfluorane, phenothiazine, and so on) should be avoided.

1411 PORPHYRIA CUTANEA PRAEMATURA DUE TO HORMONAL CONTRACEPTIVES. (Ger.) Doss, M. (Fach Klinische Biochemie im Fachbereich Humanmedizin der Universitat, Pilgrimstein 2, 3550 Marburg/Lahn, W. Germany). *Dtsch. Med. Wochenschr.* 102(23):875-877; 1977.

Porphyria cutanea tarda (PCT) was diagnosed in four patients following long-term use of hormonal preparations. PCT was due to the use of oral contraceptives in three 23- to 25-yr-old patients; two had used Eugynon for 3 and 8 yr, resp., the third, Orlest for 3 yr. PCT was also found in a 65-yr-old man who was treated with estrogens for prostate carcinoma. The genealogical investigation of one of the women revealed PCT in her father (probably manifested as a result of chronic alcoholism) and chronic hepatic porphyria type B without dermatological signs in her sister. Uroporphyrins were dominant in all patients, accounting for 51-63% of the total porphyrin, while heptacarboxyporphyrin accounted for 19-43%, hexacarboxyporphyrin for 1-7%, pentacarboxyporphyrin for <1-7%, coproporphyrin for <1-7%, and tricarboxyporphyrin for up to 1%. The very low incidence of manifest PCT compared to the large numbers of women using oral contraceptives and patients treated with estrogens suggests that a defect in uroporphyrinogen decarboxylase activity is a necessary condition of PCT. The enzymatic disturbance develops gradually from the clinically symptom-free initial stages of chronic hepatic porphyria to PCT. Differentiation between late and early forms of PCT is not justified in view of the uniform pathobiochemical findings.

1412 QUINIDINE-INDUCED HEPATITIS AND THROMBOCYTOPENIA. (Eng.) Alam, M.; Duvernoy, W. F. C.; Pickard, S. D.; Aronsohn, P. L. (Henry Ford Hosp., 2799 W. Grand Blvd., Detroit, MI 48202). *Henry Ford Hosp. Med. J.* 25(2):53-60; 1977.

The cases of four patients with quinidine-induced hepatitis are presented. Hepatitis was accompanied by malaise, anorexia, fever in two patients, and marked elevation of liver enzymes in all. Two patients had associated quinidine-induced thrombocytopenia of 81,000 and 6,600 platelets/mm^3, resp. Liver biopsy in two patients revealed small foci of hepatocellular necrosis, Kupffer cell hyperplasia, and Kupffer cells containing lipochrome. In one patient, noncaseous granulomatous lesions were seen. Symptoms subsided and laboratory tests returned to normal rapidly on withdrawal of quinidine. In one patient, drug challenge reproduced symptoms and abnormal liver function test results. Quinidine-induced hepatitis is probably more frequent than recognized heretofore and may be associated with quinidine-induced thrombocytopenia.

1413 ACUTE LEAD POISONING--AN UNUSUAL CAUSE OF HEPATITIS [Abstract]. (Eng.) Beattie, A. D.; Mullin, P.; Baxter, R.; Moore, M. R. (Southern General Hosp., Glasgow, Scotland). *Gut* 18(11):A968; 1977.

1414 HALOTHANE-RELATED HEPATITIS: CLINICAL SPECTRUM AND ROLE OF STEROID THERAPY [Abstract]. (Eng.) Varma, R. R.; Michelsohn, N. (Milwaukee County General Hosp., Milwaukee, WI). *Clin. Res.* 25(4):575A; 1977.

1415 ACETAMINOPHEN AND HEPATIC DYSFUNCTION IN INFECTIOUS MONONUCLEOSIS. (Eng.) Rosenberg, D. M.; Meyer, A. A.; Manning, I. H., Jr.; Neelon, F. A. (Natl. Heart, Lung, and Blood Inst., Bldg. 10, Room 6N26O, Bethesda, MD 20014). *South. Med. J.* 70(6):660-661; 1977.

See also, 0966, 1365, 1625, 1634.

1416 HEPATITIS A AND HEPATITIS B: DIFFERENTIAL CLINICAL, EPIDEMIOLOGICAL AND IMMUNOLOGICAL FEATURES. (Ita.) Ortana, L.; Pizzigallo, E.; Federico, G.; Camilli, G.; Laghi, V. (Istituto di Clinica Malattie Infettive, Universita Cattolica del Sacro Cuore, Rome, Italy). *Minerva Med.* 68(1): 13-28; 1977.

A study is presented of 136 patients with viral hepatitis. Radioimmunoassay of hepatitis B surface antigen (HBsAg) revealed that 58 were HBsAg positive and 78 were HBsAg negative. Mussels were responsible for the transmission of viral hepatitis in 33.33% of the HBsAg-negative group. The oral-fecal route of transmission was implicated in 85.9% of HBsAg-negative cases and 48.2% of HBsAg-positive cases. The average course of disease in hepatitis B and A, resp., was 51.7 and 36 days. Comparative determinations of the levels of transaminases, bilirubin, immunoglobulins, and complement revealed variations in behavior between two types of hepatitis. On the other hand, cellular immunity, studied by the E-rosette technique, appeared depressed in the initial phase of illness in both types of hepatitis, with return to normal upon clinical cure. Clinical cures in less than 60 days were achieved in 71.7% of the HBsAg-negative cases compared to 63.4% of the HBsAg-positive cases; indirect hyperbilirubinemia persisted in 3.7% and 6.8% of the HBsAg-negative and -positive cases, resp. Clinical cures in more than 60 days were achieved in 10.2% and 18.9% of HBsAg-negative and -positive cases, resp., with persistent indirect hyperbilirubinemia in 5.1% and 1.6% of the cases, resp. Continued elevation of transaminase levels were found in 8.9% and 6.8%, resp., of the HBsAg-negative and -positive subjects, who were dismissed from observation without further follow-up. The percentage of complete cures are about equal in the two types of hepatitis, although recovery is significantly faster in hepatitis A (HBsAg-negative hepatitis).

1417 ACUTE HEPATITIS: A PROGNOSTIC STUDY WITH OBSERVATION TIME UP TO 37 YEARS: A FOLLOW-UP STUDY OF THE IVERSEN/ROHOLM LIVER BIOPSY MATERIAL. (Eng.) Dietrichson, O.; Zoffmann, H.; Christoffersen, P.; Hilden, M.; Juhl, E.; Thomsen, A. C. (Kommunehospitalet, Copenhagen, Denmark). *Acta Med. Scand.* 202(4):271-276; 1977.

A follow-up study of 147 patients who had had hepatitis was performed 15-37 yr after the diagnostic biopsy, based on repeated liver biopsies, biochemical liver tests, autopsy reports, and death certificates. Two patients died from acute liver failure, and the development of cirrhosis was documented or strongly suspected in 22 patients. A comparison between these 24 patients and 86 patients with uncomplicated disease revealed a significantly larger number of women (79% versus 38%), a higher age (median 57 versus 40 yr), and more cases with piecemeal necrosis (83% versus 52%), confluent necrosis (54% versus 26%), and marked portal inflammation (83% versus 43%) at the time of the initial liver biopsy in the group with the poor prognosis. The results indicate that the prognosis can be determined by clinical and histological examination in patients with acute hepatitis.

1418 ACUTE VIRAL HEPATITIS IN ITALY: RESULTS OF A COLLABORATIVE STUDY. (Eng.) Guisti, G.; Ruggiero, G.; Galanti, B.; Piccinino, F.; Sagnelli, E.; Lucchelli, P. E. (Univ. Naples, 1st Medical Sch. Naples, Naples, Italy). *Acta Hepato-gastroenterol.* 24(4):226-237; 1977.

The etiological and clinical aspects of viral hepatitis were evaluated by a retrospective survey of 2,788 hospitalized Italian patients. Ninety percent of the patients had not been treated with steroids. For each case, the clinical and biochemical data were recorded, coded, computerized, and statistically analyzed. Viral hepatitis was more frequent among young patients (mean age, 26.3 yr; age range 1-75 yr; age range of highest incidence, 6-10 yr) and among men (male/female ratio, 1.26). The major factors affecting the severity of illness were age, sex, hepatitis B surface antigen (HBsAg), and transmission by blood transfusion. Both HBsAg-positive patients and older patients had deeper jaundice ($p<0.001$), longer hospitalization ($p<0.001$), higher rates of mortality, and more frequent progression to chronicity. Men had more prolonged jaundice, antigenemia, and hospitalization and a higher frequency of biochemical relapses. Furthermore, men predominated among those patients who progressed to chronicity or died of acute liver failure. In all age groups irrespective of the presence of serum HBsAg, post-transfusion hepatitis was more severe (deeper jaundice, longer hospitalization, and more frequent biochemical relapses, $p<0.01$ for all) than nontransfusion hepatitis. The death rate for all patients was 0.5% but was greater in men (0.64%), in patients over 50 yr of age (1.74%), and in transfused patients (1.58%). The lowest death rate (0.13%) was observed in children age 6-15. Age, sex, presence or absence of HBsAg, and history of blood transfusion were examined for interrelationships, and significant correlations for pairs of these parameters are given. Type B hepatitis was confirmed to be an occupational hazard for surgeons and nurses, consistent with previous observations. The need for prospective studies of viral hepatitis to permit accurate evaluation of outcome is stressed.

1419 HEPATITIS B IN WARD AND CLINICAL LABORATORY EMPLOYEES OF A GENERAL HOSPITAL. (Eng.) Levy, B. S.; Harris, J. C.; Smith, J. L.; Washburn, J. W.; Mature, J.; Davis, A.; *et al.* (128 Hollis St., Sherborn, MA 01770). *Am. J. Epidemiol.* 106(4):330-335; 1977.

A sharp increase in viral hepatitis cases, mostly type B, among the 2,000 employees of a general

hospital during 3 yr was investigated. Of the 38 cases, 22 occurred in nonphysician, ward employees. Of 189 current ward employees, 8% had antibody to hepatitis B surface antigen (anti-HBs) and 1% had hepatitis B surface antigen (HBsAg). Hepatitis B virus (HBV) seropositivity was highest for employees who worked closely with hemodialysis and renal transplant patients and for those who claimed that their ward was understaffed. Nine of the 38 cases occurred in clinical laboratory workers. Of 70 current laboratory employees, 17% were positive for anti-HBs and none for HBsAg. HBV seropositivity was highest for those working in the chemistry section (highest there among those performing blood-gas determinations and those working with the multichannel autoanalyzers) and those who routinely got blood on their skin and clothes at work. All seropositive employees worked routinely with blood. These data support the hypotheses that many hospital employees contract hepatitis B from exposure to HBsAg-positive patients and that many clinical laboratory employees contract it from exposure to HBV-contaminated blood.

1420 HEPATITIS-B VIRUS INFECTION IN ANAESTHETISTS. (Eng.) Carstens, J.; Macnab, G. M.; Kew, M. C. (Baragwanath Hosp., Johannesburg, South Africa). *Br. J. Anaesthesiol.* 49(9):887-889; 1977.

To determine whether anesthetists are at risk of developing hepatitis B virus (HBV) infection from their patients, 95 anesthetists working with black South Africans (who have a high prevalence of hepatitis B antigenemia) were questioned about attacks of viral hepatitis, and their blood was tested for hepatitis B surface antigen (HBsAg) and antibody (anti-HBs) by radioimmunoassay. Anti-HBs was detected in the sera of 17.9% anesthetists, but none was a chronic carrier of HBsAg. Two anesthetists had acute viral hepatitis during their careers, one of whom is now positive for anti-HBs. Forty-five of the anesthetists (47.4%) were known to have anesthetized patients with HBs antigenemia, and of these seven were anti-HBs-positive. Anesthetists working with a population having a high carrier rate of HBV appear to be more at risk from HBV infection than the general population.

1421 SERUM ALPHA-FETOPROTEIN LEVELS IN PATIENTS WITH ACUTE AND CHRONIC LIVER DISEASE: RELATION TO HEPATOCELLULAR REGENERATION AND DEVELOPMENT OF PRIMARY LIVER CELL CARCINOMA. (Eng.) Eleftheriou, N.; Heathcote, J.; Thomas, H. C.; Sherlock, S. (Royal Free Hosp., London NW3 2QG, England). *J. Clin. Pathol.* 30(8):704-708; 1977.

Serial serum alpha-fetoprotein determinations were carried out in 58 patients with acute liver damage to assess the value of this test as a marker of hepatocellular regeneration. One hundred sixty-three patients with chronic liver disease were also examined to determine whether this method of alpha-fetoprotein determination is of value in predicting the development of primary liver cell carcinoma. Using a radioimmunoassay technique, serum alpha-fetoprotein was detected in 50 healthy adults in concentrations of less than 20 µg/l. Among 44 patients with acute viral hepatitis, 43% exhibited a transient rise of serum alpha-fetoprotein, the peak occurring 8-9 days after the maximum recorded serum aspartate transaminase activity. Eight patients with paracetamol-induced liver injury were also shown to have transiently raised levels, the peak occurring earlier than in subjects with viral hepatitis. Six subjects with fatal fulminant hepatitis were studied; the three with the more protracted illness were noted to have increased levels before death. Twenty patients with chronic liver disease also had elevated serum alpha-fetoprotein concentrations (>20 µg/l, <500 µg/l). In four, primary liver cell cancer developed; in two of these the serum alpha-fetoprotein levels rose progressively, and in two it remained raised but at low levels. The observations in the patients with paracetamol-induced liver injury suggest a correlation between the degree of liver cell necrosis and subsequent alpha-fetoprotein production. The increase in serum alpha-fetoprotein in fulminant hepatitis may be related to the duration of illness regardless of the final outcome. Steadily increasing levels are highly suspicious of the development of primary liver cell carcinoma in patients with chronic liver disease.

1422 HBAg-POSITIVE CHRONIC LIVER DISEASE ASSOCIATED WITH CIRRHOSIS AND HEPATOCELLULAR CARCINOMA IN THE SENOI. (Eng.) Sumithran, E.; Prathap, K. (Dept. Pathology, Univ. Malaya, Kuala Lumpur, Malaysia). *Cancer* 40(4):1618-1620; 1977.

To confirm the high incidence of primary hepatocellular carcinoma (PHC) among the Senoi, a Malaysian aborigine group, all tumors diagnosed by histologic examination of surgical and necropsy material from a local hospital over a 13-yr period were reviewed, and the presence of hepatitis B surface antigen (HBsAg) was assessed in paraffin sections of all livers from the necropsied cases. PHC was present in 10 of 22 Senoi patients with cirrhosis. All 22 livers contained hepatocytes that stained with Shikata's orcein stain and specific immunoperoxidase and immunofluorescent stains for HBAg. This observation raises the strong possibility that hepatitis B may be an important etiologic factor in the development of cirrhosis and PHC in the Senoi. The reason for the high susceptibility of the Senoi for hepatitis B virus infection is not clear, and the role of aflatoxin in the pathogenesis of PHC in the Senoi has yet to be determined. That the Senoi are a numerically small community, maintaining their own unique dietary and social customs and living in readily accessible areas in the Malaysian jungle, makes them an ideal population for the study of factors in the etiology of liver cancer.

1423 HEPATITIS-ASSOCIATED LIPID STORAGE MYOPATHY. (Eng.) Patten, B. M.; Shabot, J. M.; Alperin, J.; Dodson, R. F. (Baylor Coll. Medicine, Houston, TX 77030). *Ann. Intern. Med.* 87(4):417-421; 1977.

The case of a 23-yr-old woman who developed severe myalgias in association with mild hepatitis B surface antigen-positive hepatitis is presented. Muscle biopsy showed myriads of microvacuoles filled with neutral lipid. Prednisone therapy (40 mg/day) was associated with complete clearing of all clinical and laboratory abnormalities. Since muscle carnitine levels were normal before treatment, it is concluded that infection with hepatitis virus may induce noncarnitine-deficient lipid storage myopathy in man.

1424 THE LIVER DURING AND AFTER FULMINANT HEPATITIS. (Eng.) Horney, J. T.; Galambos, J. T. (Emory Univ. Sch. Medicine, 69 Butler St., S.E., Atlanta, GA 30303). *Gastroenterology* 73(4): 639-645; 1977.

To study changes in liver histology during and after fulminant hepatitis, biopsies were obtained 5 to 14 days after onset of grade 3 or 4 coma, in 15 consecutive patients who recovered from fulminant hepatitis. In nine patients, follow-up biopsy was obtained 6 to 60 months after acute hepatitis, and autopsy was performed in two patients who died in 4 months from complications of hepatitis (aplastic anemia) or of corticosteroid therapy (sepsis). During fulminant illness, the biopsy findings were multilobular necrosis in four patients, confluent (bridging) necrosis in nine, and only portal inflammation in one. The duration or the grade of coma did not correlate with the severity of necrosis on the biopsy. Follow-up biopsy showed development of chronic (active) hepatitis in three of nine patients (with cirrhosis in one of these). Chronic liver disease was not found in the two autopsies. If fulminant hepatitis is the result of vigorous cell-mediated immune attack on hepatocytes, then this process cannot always eradicate chronic hepatitis B surface antigenemia, nor can it always prevent the development of chronic (active) hepatitis or cirrhosis.

1425 LIVER-SPECIFIC PROTEINS AND AUTOIMMUNITY IN INFLAMMATORY LIVER DISEASE. (Ita.) Alberti, A.; Realdi, G.; Bortolotti, F.; Trevisan, A.; Tremolada, F.; Rigoli, A. (Istituto di Medicina Clinica, Cattedra di Patologia Medica dell'Universita degli Studi di Padova, Padua, Italy). *Boll. Ist. Sieroter. Milan.* 55(5):411-425; 1976.

Cellular immune reactions against liver-specific proteins (LP_1) were studied by the leukocyte migration inhibition test in patients with different kinds of liver disease. Sixty-six percent of patients with chronic active hepatitis showed cellular immunity to LP_1. There was no difference in reactivity in patients who were serum positive for hepatitis B antigen (HBAg) and those who were HBAg-negative. Fifty-seven percent of patients with viral hepatitis B who tested in the acute phase of the disease had immunity to LP_1. After 6-12 months, only 17.8% of these same patients were still LP_1 positive Inhibition of leukocyte migration appears to decrease in these patients as virus is cleared from the liver. Inhibition of leucocyte migration was also found in 80% of HBAg-positive patients with chronic persistent hepatitis and in 23% of asymptomatic carriers of HBAg, most of whom progressed to cases of chronic active hepatitis. The cell-mediated immune response appears to play a major role in the pathogenesis of chronic active hepatitis. Infection with the hepatitis B virus must also be considered as a possible cause of autoimmune liver disease.

1426 LYMPHOCYTE POPULATIONS IN HBAg-POSITIVE ACUTE HEPATITIS. (Ita.) Bianchi, F. B.; Zauli, D.; Marchesini, G.; Gritti, F. M.; Fabbri, L.; Pisi, E. (Istituto Patologia Medica, Universita di Bologna, Bologna, Italy). *Minerva Gastroenterol.* 22(3):251-252; 1976.

T and B lymphocyte levels were determined in 11 patients with acute hepatitis and 21 patients with chronic uremia who were being treated with renal dialysis. All of the acute hepatitis patients and 11/21 uremia patients were seropositive for hepatitis B antigen (HBAg). There was a significant reduction in the numbers of circulating lymphocytes and monocytes in all HBAg-positive patients. The absolute numbers and the percentage of circulating B lymphocytes increased from 674 ± 120 cells/cm^3 or 27.0 ± 4.0% in controls to 885 ± 303 cells/cm^3 or 42.7 ± 9.6% in hepatitis patients; the increase in absolute number was not significant, whereas the percentage increase was significant ($p<0.001$). The absolute number and percentage of circulating T lymphocytes decreased significantly in all patients, especially in HBAg-positive uremia patients in whom values fell from 1,567 ± 428 cells/cm^3 or 65.5 ± 8.0% in the controls to 844 ± 434 cells/cm^3 or 45.6 ± 15.3%. The lymphopenia that occurs in the early stages of viral hepatitis is the result of a reduction in the number of circulating T lymphocytes. This reduction in the uremia patients is a reflection of the inhibitory action of uremia on the immune response.

1427 HUMORAL AND CELLULAR IMMUNE REACTIONS AGAINST HBsAg AND AGAINST LIVER-SPECIFIC PROTEINS IN THE COURSE OF ACUTE AND CHRONIC HEPATITIS. (Ita.) Realdi, G.; Alberti, A.; Bortolotti, F.; Tremolada, F.; Diodati, G.; Cadrobbi, P. (Clinica Medica Generale dell'Universita di Padova, Padova, Italy). *Minerva Gastroenterol.* 22(3):207-212; 1976.

Cell-mediated immunity against liver specific protein (LSP) and hepatitis B surface antigen (HBsAg) was studied by means of the leukocyte migration inhibition test in 80 patients and 20 control subjects. Twenty-two patients had acute viral hepatitis B (AVHB), 12 had chronic persistent hepatitis (CPH), 15 had chronic active hepatitis (CAH) and were seropositive for hepatitis B antigen (HBAg), 16 had CAH and were HBAg-negative, and 15 had alcoholic cirrhosis. LSP was found in 54%, 53%, 75%, and 16% of patients with AVHB, (HBAg-positive) CAH, (HBAg-negative) CAH, and CPH, resp. Cirrhotic patients and controls were LSP-negative. In CAH patients treated with prednisone, there was a reduction in the autoimmune response, while untreated patients remained

LSP-positive over a 12-month period. Although 50% of AVHB patients were LSP-positive in the acute phase of their disease, only 10% were still positive 6-12 months later. Thirty-one percent of HBAg-positive CAH patients exhibited cellular immunity to HBsAg. In the acute disease phase, 51% of AVHB patients had cell-mediated immunity to HBsAg; cellular immunity appeared in 83% after 3 months and in 58% after 12 months. Antibody (Ab) to HBsAg in these patients was low in the acute phase, but climbed steadily from 3-12 months, by which time 80% were HBsAb positive. The autoimmune reaction to LSP in AVHB patients is most likely a result of membrane protein alteration by the virus. Sensitivity to LSP is limited and disappears with the clearance of the virus from the body. In HBAg-positive CAH patients, the virus persists in the body, and autoimmunity to LSP persists also. HBAg-negative CAH is a classic autoimmune disease, which may be initiated by viruses, drugs, or other external agents; cell-mediated immunity to HBsAg in this disease suggests previous contact with the HB virus in some patients. It is also suggested that an element of genetic predisposition to this autoimmune disease may exist.

1428 CORRELATION BETWEEN TITER OF ANTIBODY TO HEPATITIS B CORE ANTIGEN AND PRESENCE OF VIRAL ANTIGENS IN THE LIVER. (Eng.) Kojima, M.; Udo, K.; Takahashi, Y.; Yoshizawa, H.; Tsuda, F.; Itoh, Y.; *et al.* (Gifu Prefectural Hosp., Gifu-ken, Japan). *Gastroenterology* 73(4):664-667; 1977.

In an effort to find a reliable indicator of persistent hepatitis B virus (HBV) infection, markers of HBV infection in the serum were correlated with the presence of both hepatitis B core antigen (HBcAg) and hepatitis B surface antigen (HBsAg) in the livers of 31 autopsied patients with liver cirrhosis. Liver specimens were stained for HBcAg and HBsAg by the direct immunofluorescence method. All specimens had detectable levels of antibody to HBcAg (anti-HBc). Their premortem serum samples were tested for HBsAg, antibody to HBsAg (anti-HBs), and anti-HBc. Persistent (HBV) infection, as judged by circulating and/or liver hepatitis B antigens, was identified in 18 patients, and all of them revealed a high titer of anti-HBc ranging from 2^{11} to 2^{16} by the immune adherence hemagglutination method. In contrast, the anti-HBc titer of the remaining 13 patients without detectable HB antigens, was less than 2^{9}, and the geometric mean titer of anti-HBc of the patients with persistent HBV infection was significantly higher than that of the patients without (13.9 ± 1.55 versus 7.23 ± 1.30; t test, $p<0.001$). A combination of circulating anti-HBs and hepatic HB antigens was found in one patient, whose serum revealed an anti-HBc titer of 2^{12}. A high titer of anti-HBc in the serum (immune adherence hemagglutination titer $\geq 2^{11}$) seems to be a reliable indicator of persistent HBV infection in the liver.

1429 EXPRESSION OF HEPATITIS B VIRUS-SPECIFIC MARKERS IN ASYMPTOMATIC HEPATITIS B SURFACE ANTIGEN CARRIERS. (Eng.) Hess, G.; Arnold, W.; Shih, J. W. K.; Kaplan, P. M.; Purcell, R. H.; Gerin, J. L.; *et al.* (Second Dept. Internal Medicine, Univ. Mainz, 6500 Mainz, W. Germany). *Infect. Immun.* 17(3):550-554; 1977.

The state of hepatitis B virus infection was assessed in 22 asymptomatic hepatitis B surface antigen (HBsAg) carriers. The presence of serum hepatitis B e antigen (HBeAg) was closely associated with serum HBsAg-specific DNA polymerase activity, hepatitis B core antigen (HBcAg) in serum and liver cell nuclei, and a histological picture of chronic hepatitis. No HBsAg-specific DNA polymerase activity or HBcAg was detected in highly concentrated sera positive for antibody to HBcAg. In addition, liver biopsy specimens from carriers with antibody to hepatitis B e antigen (anti-HBe) were negative for HBcAg by immunofluorescence, and the liver histology was either normal or revealed only fatty changes. These data indicate that the anti-HBe-positive sera contained either no Dane particles or, if present, at least a 500-fold lower concentration of Dane particles than that found in HBeAg-positive sera.

1430 A DIRECT IMMUNOFLUORESCENCE METHOD FOR THE DETECTION OF HEPATITIS B CORE ANTIGEN IN FORMALIN-FIXED AND GELATIN-EMBEDDED LIVER SPECIMENS. (Eng.) Yoshizawa, H.; Itoh, Y.; Akahane, Y.; Tsuda, F.; Miyakawa, Y.; Mayumi, M. (Tokyo Metropolitan Inst. Medical Science, Bunkyo-ku Tokyo 113, Japan). *J. Clin. Pathol.* 30(8):776-778; 1977.

A simple method for detecting hepatitis B core antigen (HBcAg) in formalin-fixed liver specimens by direct immunofluorescence is reported. Liver tissue was obtained at necropsy from patients with various hepatic diseases and was fixed and stored in 10% formalin. The specimens are washed free of formalin in tap water for 12 hr and incubated in distilled water containing 0.01% NaN_3 and 5% gelatin at 37 C for 12 hr, and successively in water supplemented with 10% gelatin at 37 C for 4 hr. The specimens are transferred to a Petri dish filled with 10% gelatin at 37 C, and the dish is allowed to consolidate at room temperature. The specimens are then snap-frozen in n-hexane. In one specimen from a patient with liver cirrhosis, HBcAg-positive immunofluorescence was exclusively localized in the nucleus. About 80% of the hepatocytes were positive with a clear bright fluorescence and virtually no background staining. The staining of hepatitis B surface antigen (HBsAg) was localized in the cytoplasm, and no nuclear fluorescence was observed. The detectability of both HBcAg and HBsAg by the direct immunofluorescence method was similar in fixed and unfixed specimens embedded in gelatin. The basis for the success of this method is apparently the embedding process, with which the dehydration process for embedding in paraffin is avoided.

1431 SOLID PHASE RADIOIMMUNOASSAY FOR QUANTITATING ANTI-HBs IN HEPATITIS B IMMUNE

GLOBULIN. (Eng.) Ratliff, E. A.; Pollack, W.; Tripodi, D.; Ho, T. (Ortho Res. Inst. Medical Sciences, Raritan, NJ 08869). *Int. Arch. Allergy Appl. Immunol.* 54(3):231-237; 1977.

A solid-phase radioimmunoassay technique was modified to quantitate the antibody in hepatitis B immune globulin. This procedure allows the data to be resolved into a Sips adsorption isotherm. This treatment of data permits the total antibody, intrinsic average binding constant, and index of heterogeneity to be determined, since it was demonstrated that the antibody behaves as if it is monovalent with respect to binding its antigen. Two assay procedures based upon different methods of measuring nonspecific binding are described. In the first, a labeled control globulin known to be free of antibody to hepatitis B surface antigen is used. In the second assay procedure, buffer-treated tubes are used as the "negative" control. The methods were found to give indistinguishable results, but the authors believe that the latter will offer the greatest utility.

1432 PRESENCE OF HBsAg AND IgG ON THE LIVER CELL MEMBRANE IN PATIENTS WITH ACUTE AND CHRONIC HEPATITIS. (Ita.) Alberti, A.; Realdi, G.; Tremolada, F.; Rigoli, A. M.; Fattovich, G.; Spina, G. P. (Clinica Medica Generale, Univ. Padova, Padua, Italy). *Minerva Gastroenterol.* 22(3):235-240; 1976.

The presence of the hepatitis B antigen (HBAg) and IgG on the surface of hepatocytes from patients with acute and chronic hepatitis was determined by immunofluorescence. Ten patients with acute hepatitis B were examined during the first week of illness. Seven of these 10 had HBAg at the cell surface and 5/10 had intracellular HBAg. An additional 13 patients with acute hepatitis B were examined during the recovery phase of the disease. None of these patients had HBAg on the cell surface, but 33% of them had intracellular HBAg. Five patients with chronic persistent hepatitis B were studied also. Two of these five were seropositive for HBAg. One of the seropositive patients had intracellular HBAg. Fifteen patients with chronic active hepatitis (CAH) were also studied. Eight patients were seropositive and seven were seronegative for HBAg. All of the positive patients had HBAg located within the cell but not at the cell surface. None of the seronegative CAH patients had intracellular IgG. Surface IgG was found in 10% of the acute hepatitis B patients examined in the first week of the study and 7.6% of these examined in the recovery phase; 75% of the seropositive and 57% of the seronegative CAH patients also had surface IgG. All of the other patients were negative for surface IgG. Five patients with alcoholic cirrhosis and 4 normal controls were negative for both HBAg and IgG at both locations. These results suggest that in patients with acute hepatitis B, cellular immune reactions are perhaps responsible for cell lysis and the elimination of both infected cells and the antigenic organism. In CAH patients, cellular antibodies are probably directed against diverse antigens.

1433 e ANTIGEN AND ANTIBODY IN OUTBREAKS OF HEPATITIS B IN TWO RENAL DIALYSIS UNITS. (Eng.) Gibson, P. E. (Central Public Health Lab., Colindale Ave., London NW9 5HT, England). *J. Clin. Pathol.* 30(8):717-719; 1977.

In two renal dialysis units that had outbreaks of hepatitis B, patients were studied for the presence of e antigen and antibody. In one unit, in which the illness was mild and most of the 27 patients (78%) became carriers of hepatitis B surface antigen (HBsAg), all but one patient had e antigen. Most of the HBsAg carriers had persisting e antigen, and most of the transient patients had also transient e antigen. In the other unit, in which the illness was more severe, none of the 10 patients became a carrier. Only 30% had e antigen during the acute phase of illness and 40% developed anti-e just before clearing HBsAg. The differences between these two units may reflect strain differences in the infectivity of hepatitis B virus.

1434 HEPATITIS B e ANTIGEN AND ANTIBODY ACTIVITY IN HEPATITIS B VIRUS INFECTION. (Eng.) Tiku, M. L.; Makhdoomi, G. M.; Beutner, K. R.; Nath, N.; Ogra, P. L. (Children's Hosp., 219 Bryant St., Buffalo, NY 14222). *J. Pediatr.* 91(4): 540-544; 1977.

Groups of institutionalized subjects, volunteer blood donors with serologic evidence of asymptomatic hepatitis B virus infection, and patients with acute type B viral hepatitis were studied for the presence of hepatitis B e antigen (HBeAg) and antibody (anti-HBe) in a sequential manner over 2-3 yr. HBeAg was detected in 9.5% of 226 institutionalized residents and 105 volunteer blood donors and in 24% of 46 patients with acute type B viral hepatitis. HBeAg-positive subjects frequently had persistently elevated SGPT levels. Anti-HBe activity was observed in 26% to 32% of subjects positive for hepatitis B surface antigen (HBsAg). Long-term follow-up indicated that HBsAg-positive subjects with anti-HBe did not eventually become seronegative for HBsAg.

1435 EVALUATING THE SEVERITY OF VIRAL HEPATITIS USING A COAGULOGRAM. (Rus.) Sorinson, S. N.; Zhdakova, I. N. (Gorki Medical Inst., Gorki, USSR). *Sov. Med.* (5):27-30; 1977.

1436 HEPARIN IN THE TREATMENT OF SEVERE FORMS OF VIRAL HEPATITIS. (Rus.) Pateyuk, V. G.; Fedyanina, Yu. P. (Chita Medical Inst., Chita, USSR). *Sov. Med.* (5):33-36; 1977.

1437 MEASURES TO CONTROL VIRAL HEPATITIS. (Rus.) Stepanov, G. P.; Bondarenko, M. P. (Central Scientific Res. Inst. Epidemiology, Ministry Health USSR, Moscow, USSR). *Sov. Med.* (5):125-129; 1977.

1438 METHOD FOR THE IDENTIFICATION OF AUSTRALIA ANTIGEN USING THE COUNTER IMMUNOELECTROPHORESIS INHIBITION TEST. (Rus.) Gelemey, V. P. (Lvov Medical Inst., Lvov, USSR). *Lab. Delo* (6):353-356; 1977.

1439 INVESTIGATION OF BLOOD SERUM PROTEINS IN PATIENTS WITH ACUTE VIRAL HEPATITIS USING THE METHOD OF DISC ELECTROPHORESIS IN POLYACRYLAMIDE GEL. (Rus.) Levina, L. D.; Ambalov, Yu. M.; Kartashev, V. V. (Rostov Medical Inst., Rostov, USSR). *Klin. Med.* 55(2):126-131; 1977.

1440 AUSTRALIA ANTIGEN AND VIRAL HEPATITIS: CLINICAL AND STATISTICAL CONSIDERATIONS. (Ita.) Menicagli, V.; Pardelli, G. C. (Spedali Riuniti, Livorno, Italy). *Minerva Med.* 68(12): 769-772; 1977.

1441 SHORT ALTERNATING CORTICOTHERAPY WITH SINGLE DOSES AT 48-HOUR INTERVALS IN ACUTE VIRAL HEPATITIS. (Rum.) Caruntu, F.; Angelescu, C.; Predoviciu, F. (Spitalul clinic de boli infectioase "Colentina," Bucharest, Rumania). *Med. Interna* 28(3):205-210; 1976.

1442 REACTION OF ALLERGENOLEUKOCYTOLYSIS IN VIRAL HEPATITIS (SERUM HEPATITIS). (Rus.) Pyntya, V. E. (Kishinev Medical Inst., Kishinev, USSR). *Ter. Arkh.* 48(12):45-48; 1976.

1443 MANAGEMENT OF TYPE B VIRAL HEPATITIS. (Fre.) Rousseau, C. (Centra Hospitalier de l'Universite Laval, 2705, boul. Laurier, Quebec G1V 4G2, Canada). *Union Med. Can.* 106(3):351-355; 1977.

1444 SIGNIFICANCE OF AUSTRALIA ANTIGEN IN THE DIFFERENTIAL DIAGNOSIS AND PROGNOSIS OF HEPATITIS. (Ger.) Baenkler, H. W. (Institut und Poliklinik fur Klinische Immunologie der Universitat, Krankenhausstrasse 12, 852 Erlangen, W. Germany). *Fortschr. Med.* 95(9):577-579; 1977.

1445 PERIPHERAL BLOOD LYMPHOCYTE POPULATIONS IN VIRAL HEPATITIS. (Pol.) Celinska-Szpytko, E.; Wolko, K.; Mackiewicz, S. (Acad. Medicine, Poznan, Poland). *Pol. Arch. Med. Wewn.* 56(2/8):113-118; 1976.

1446 EMETINE IN THE TREATMENT OF VIRAL HEPATITIS. (Cze.) Kosina, F.; Truksova, D. (Krajska nemocnice, 400 43 Usti nad Labem, Czechoslovakia). *Cas. Lek. Cesk.* 115(47):1454-1457; 1976.

1447 PATHOGENETIC MECHANISMS OF HYPERBILIRUBINEMIA IN WOMEN WITH VIRAL HEPATITIS. (Rus.) Yatsenko, L. A. (Medical Inst., Ordzhonikidze, USSR). *Sov. Med.* (5):30-32; 1977.

1448 HEPATITIS B ANTIGEN IN THE DIAGNOSIS AND COURSE OF VIRAL HEPATITIS IN PREGNANT WOMEN. (Ger.) Boron, P.; Bobrowska, E.; Boron-Kaczmarska, A.; Grabianska, A.; Karwowska, K.; Szpakowicz, T.; Rzewnicki, I. (Medizinischen Akademie in Bialystok, VR Polen, ul. Zurawia 14, 15-540 Bialystok, Poland). *Dtsch. Gesundheitsw.* 32(28):1310-1312; 1977.

1449 EPIDEMIOLOGICAL AND CLINICAL ARGUMENTS CONCERNING THE PROGNOSIS OF VIRAL HEPATITIS. (Fre.) Pariente, E.; Laverdant, C. (Clinique Medicale B, H.I.A. Begin, 69 avenue de Paris, F 94160 Saint-Mande, France). *Med. Armees* 4(2):105-110; 1976.

1450 DIAGNOSTIC COEFFICIENTS FOR DIFFERENTIATING INFECTIOUS AND SERUM HEPATITIS. (Rus.) Titov, M. B.; Gerasun, B. A.; Grinbaum, A. S.; Germaniuk, I. L. (Lvov Medical Inst., Lvov, USSR). *Vrach. Delo* (5):146-150; 1977.

1451 ANTI-GAMMAGLOBULIN FACTORS, IMMUNOGLOBULIN M, AND AUSTRALIA ANTIGEN IN ACUTE AND CHRONIC ACUTE VIRAL HEPATITIS. (Ita.) Tigano, F.; Ferlazzo, B.; Barrile, A.; Caruso, R. (Via Risorgimento, 172, 98100 Messina, Italy). *Prog. Med.* 32(14):562-568; 1976.

1452 SYNDROME OF NEONATAL HEPATITIS [Abstract]. (Eng.) Licastro, R.; Ciocca, M. (No affiliation given). *Arq. Gastroenterol.* 14(3): 160-161; 1977.

1453 ACUTE HEPATITIS--CLINICAL, HUMORAL AND HYSTOLOGIC [sic] COURSE [Abstract]. (Eng.) Licastro, R.; Ciocca, M.; Ramonet, M.; Costa, J. A. (No affiliation given). *Arq. Gastroenterol.* 14(3): 162; 1977.

1454 DISTURBED CELL-MEDIATED IMMUNITY IN LIVER DISEASE: CAUSE OR EFFECT [Abstract]? (Eng.) Cowlishaw, J. L.; Smith, C. I.; Bui, C. T.; Cooksley, W. G. E.; Campbell, C. B.; Halliday, J. W.; Powell, L. W. (Dept. Medicine, Univ. Queensland, Brisbane, Australia). *Clin. Exp. Pharmacol. Physiol.* 4(5):465-466; 1977.

1455 THE PATHOGENESIS OF URTICARIA ASSOCIATED WITH ACUTE VIRAL HEPATITIS TYPE B [Ab-

stract]. (Eng.) Dienstag, J. L.; Rhodes, A. R.; Wands, J. R.; Bhan, A. K.; Dvorak, A. M.; Isselbacher, K. J. (Massachusetts General Hosp., Boston, MA). *Gastroenterology* 73(5):1217; 1977.

1456 NATURAL HISTORY OF HEPATITIS B SURFACE ANTIGEN (HBsAg) CHRONIC CARRIER STATE [Abstract]. (Eng.) Childress, L.; Sampliner, R. E. (Div. Gastroenterology, Univ. Maryland, Baltimore, MD). *Gastroenterology* 73(5):1216; 1977.

1457 PREVALENCE OF HEPATITIS B (HB) INFECTION AMONG HOUSEHOLD CONTACTS OF CHRONIC HEPATITIS B SURFACE ANTIGEN (HBsAg) CARRIERS [Abstract]. (Eng.) Bernier, R.; Sampliner, R. E. (Johns Hopkins Sch. Hygiene, Baltimore, MD). *Gastroenterology* 73(5):1213; 1977.

See also, 0956, 1106, 1141, 1142, 1365, 1366, 1380, 1392, 1403, 1415, 1466, 1467, 1481, 1634.

LIVER AND BILIARY TRACT

Chronic Hepatitis

1458 CLINICAL SIGNIFICANCE OF SERUM COMPLEMENT IN CHRONIC LIVER DISEASE AND ACUTE HEPATIC INSUFFICIENCY. (Ita.) Naccarato, R.; Farini, R.; Chiaramonte, M.; Meani, A.; Giordano, C.; Gritti, G. (Clinica Medica I, Universita degli Studi, Padova, Italy). *Minerva Gastroenterol.* 22(3):213-218; 1976.

Serum levels of two complement fractions, C3 and C4, were determined in a series of patients with various acute and chronic liver diseases. The C3 fraction of complement was much lower in patients with massive liver necrosis, chronic active hepatitis (CAH) with cirrhosis, cirrhosis with and without ascites, and cirrhosis after portacaval anastomosis than in normal controls. In patients with primary bilateral cirrhosis (CPB) however, C3 levels were much higher than control values. In patients with CAH without cirrhosis, C3 levels were not appreciably different from the control levels. In CAH patients, there was no correlation between C3 levels and serum levels of antibodies and immunoglobulins but there was a significant correlation between C3 levels and pseudocholinesterase levels. In patients with CPB, there was a correlation between C3 levels and alkaline phosphatase levels but not between C3 levels and bilirubinemia. There was no correlation between C3 levels and any immunological indicators. C4 levels paralleled C3 levels in all patients. There was no evidence that complement was used in the formation of immunological components in the blood. It is concluded that C3 and C4 levels cannot be used to follow the immune response in CAH patients and that the significance of C3 and C4 levels in CPB patients cannot be determined from these studies.

1459 EVALUATION OF CELLULAR IMMUNITY BY SKIN TEST IN CHRONIC HEPATITIS AND LIVER CIRRHOSIS. (Ita.) Savorani, G.; Sarti, G. (Ospedale Provinciale Specializzato M. Malpighi, Bologna, Italy). *Boll. Soc. Ital. Biol. Sper.* 52(12):859-865; 1976.

Cellular immunity in terms of sensitivity to dinitrochlorobenzene (DNCB) and reaction to five intradermal skin tests were measured in 6 patients with chronic aggressive hepatitis (CAH), 9 patients with chronic persistent hepatitis (CPH), and 45 patients with cirrhosis. Twelve of the cirrhosis patients were classified as "compensated" and 33 were classified as "noncompensated." Eight of 9 CPH patients, 4 of 6 CAH patients, 8 of 12 "compensated" cirrhosis patients, and 9 of 23 "noncompensated" cirrhosis patients were DNCB-positive. Nine of 9 CPH patients, 5 of 6 CAH patients, 12 of 12 "compensated" cirrhosis patients, and 24 of 33 "noncompensated" cirrhosis patients were positive for at least one of the five intradermal tests. All 29 DNCB-positive patients were also positive for at least one intradermal test. Of the 31 DNCB-negative patients, 21 were positive for at least one intradermal test while 10 were both DNCB negative and intradermal test negative. Of these 10 patients, 8 were "noncompensated" cirrhosis patients and one was a CAH type B patient. Cellular immunity remains generally intact in CPH patients and in patients with moderate cases of CAH. Cirrhosis patients first lose the ability to respond to new antigens and then the capacity to recognize an antigen with which they have had previous contact. There appears to be a correlation between a deficiency in cellular immunity and the type and phase of liver disease. DNCB appears to be a more sensitive indicator of this deficiency than are intradermal skin tests.

1460 AUTOIMMUNITY IN CHRONIC ACTIVE HEPATITIS AND DIABETES MELLITUS. A REVIEW. (Eng.) Galbraith, R. M.; Fudenberg, H. H. (Medical Univ. South Carolina, Charleston, SC 29401). *Clin. Immunol. Immunopathol.* 8(1):116-149; 1977.

Current evidence for the occurrence of autoimmunity in chronic active hepatitis and diabetes mellitus is reviewed. In addition, factors of potential importance in the initiation and perpetuation of these potentially damaging reactions are discussed in relation to the genetics of these disorders. The development of techniques for examining cellular immunity, particularly the leukocyte migration test, made the search for evidence of organ-specific immunity in chronic active hepatitis possible. Leukocyte inhibitory factor production has been measured in most studies, but a number of different antigens have been used. The major difficulty in these studies has been in establishing vital cultures of normal adult hepatocytes, although diseased liver tissue from patients with alcoholic liver disease has been successfully established in long-term culture. Progress in the investigation of humoral immunity to lipoprotein derived from the cell membrane (LP-I) has lagged behind that of cellular responses, since the demonstration of liver-specific antibodies in the serum of patients with chronic active hepatitis is more difficult. One investigator has used a passive hemagglutination technique to detect antibodies to liver-specific protein, the antigens used being a mixture of LP-I and a smaller cytoplasmic antigen, LP-II. Human studies demonstrating evidence of organ-specific autoimmunity in chronic active hepatitis have been reinforced by a number of animal experiments. In one case, chronic hepatitis was successfully induced in rabbits by means of repeated injections of human and rabbit liver-specific protein in complete Freund's adjuvant. The accumulation of evidence of autoimmune reactions in diabetes mellitus has in many respects run parallel to that in chronic active hepatitis. Antibodies directed against crude homogenates of pancreas including exocrine tissue were first described in 1960.

1461 HLA AND CHRONIC ACTIVE HEPATITIS. (Eng.) Mazzilli, M. C.; Trabace, S.; DiRaimondo, F.; Visco, G.; Gandini, E. (Cattedra di Genetica Medica, Universita degli Studi di Roma, Ospedale L. Spallanzani, Via Portuense, 292 Roma, Italy). *Digestion* 15(4):278-285; 1977.

Histocompatibility antigen (HLA) typing (24 alleles of the A and B loci) was performed in 42 patients with chronic active hepatitis (CAH). The criteria used for diagnosis were (a) histological profile of CAH, (b) SGOT transaminase levels above 50 mU/ml, (c) serum γ-globulin above 2 g% or at least two positive immunologic tests, and (d) clinical course lasting at least 180 days or cirrhosis evident at biopsy. An increased frequency of HLA-A3 was observed (47.6% compared to 19.1%) in 266 healthy controls. The phenotype association A3/Bw35 also was increased (28.5% in the CAH groups compared to 6.0% in controls). HLA typing was performed in seven families of seven A3/Bw35 CAH patients. In five of the families, the A3/Bw35 haplotypic segregation was proven. The data in this study indicate that the HLA phenotype is a marker of a dominant susceptibility to the disease.

1462 HLA IN DIFFERENT FORMS OF CHRONIC ACTIVE HEPATITIS. A COMPARISON BETWEEN ADULT PATIENTS AND CHILDREN. (Eng.) Freudenberg, J.; Baumann, H.; Arnold, W.; Berger, J.; Meyer zum Buschenfelde, K. H. (Blutspendedienst des DRK-Landesverbandes Rheinland-Pfalz Burgweg 5-7, D-6550 Bad Kreuznach, Mainz, W. Germany). *Digestion* 15(4): 260-270; 1977.

The presence or absence of hepatitis B surface antigen (HBsAg) and autoantibodies (AutoAb) were measured and correlated with histocompatibility antigens (HLA) in 108 adult patients and 34 children with chronic active hepatitis (CAH). All patients had the morphological profile typical of CAH and clinical and biochemical signs of high inflammatory activity of at least 6-months duration. Patients were divided into four groups: (1) AutoAb-positive and HBsAg-negative (probably causing an autoimmune type of CAH), (2) AutoAb-negative and HBsAg-negative (pathogenic mechanism unknown but may be a nonhepatitis B virus), (3) HBsAg-positive and AutoAb-negative, and (4) HBsAg-positive and AutoAb-positive (most likely caused by hepatitis B virus). The distribution of HLA among the four groups showed no statistically significant differences. When HLA levels in these groups were compared with controls, an increased frequency of HLA-B8 and HLA-A_1 was noted in the autoimmune type of CAH (group 1). The relative risk for HLA-B8-positive adults to get an autoimmune hepatitis is 19.6, compared to 7.0 in children, and 15.4 in combined groups. No other significant deviation of HLA antigens for the different types of CAH was observed.

1463 CHANGES IN LABORATORY PARAMETERS IN LIVER DISEASE PATIENTS AFTER TREATMENT WITH TOXEPASE COMPLEX. (Ita.) Lenzi, S. (I Istituto di Patologia Speciale Medica e Metodologia Clinica, Universita di Bologna, Bologna, Italy). *Minerva Med.* 67(46):2961-2974; 1976.

Twenty patients with liver disease (9 with chronic aggressive hepatitis [CAH], 2 with chronic persisting hepatitis [CPH], and 9 with cirrhosis) were treated with Toxepase complex, which consists of uridine-5-diphosphoglucose, glutathione, and vitamin B_{12}. Treatment was evaluated by determining LDH, glutamic acid dehydrogenase, alkaline phosphatase, γ-glutamyl transpeptidase, and nonspecific cholinesterase activities. These enzyme activities can indicate cellular necrosis, cholestasis, and impaired protein synthesis. Two weeks after the initiation of treatment, the serum levels of these enzymes decreased, indicating a return to normal liver function in the patients. This return to normal levels occurred in all patients and was most apparent in those with CAH and cirrhosis. CPH patients benefited least from treatment. Toxepase complex appears to be useful in the treatment of CAH and liver cirrhosis.

1464 NEW CONCEPTS ON THE PATHOGENESIS OF THE HEPATORENAL SYNDROME [Abstract]. (Eng.)

Gentilini, P.; Laffi, G.; Buzzelli, G.; Cappelli, G.; Stefani, P.; Paladini, S.; Arcangeli, A. (III Dept. Medical Pathology, Univ. Florence, Florence, Italy). *Gastroenterology* 73(5):1221; 1977.

1465 STEROID TREATMENT OF HBsAg-POSITIVE SEVERE CHRONIC ACTIVE LIVER DISEASE (CALD): DETERMINANTS OF EARLY PROGNOSIS [Abstract]. (Eng.) Czaja, A. J.; Wolf, A. M. (Mayo Clinic, Rochester, MN). *Gastroenterology* 73(5):1217; 1977.

1466 CHRONIC ACTIVE HEPATITIS IN A CHILD WITH HEMOPHILIA A [Letter to Editor]. (Eng.) Reaman, G.; Stachewitsch, A.; Mishkin, S. (Montreal Children's Hosp., Montreal, Quebec, Canada). *Can. Med. Assoc. J.* 117(7):731-732; 1977.

1467 CLINICAL SIGNIFICANCE OF THE ORCEIN STAIN [Abstract]. (Eng.) Whitcombe, F. F., Jr.; Haggitt, R. C.; Tornay, A. S., Jr.; Kutteruf, G. C. (Lahey Clinic Foundation, Boston, MA). *Gastroenterology* 73(5):1254; 1977.

1468 C1q BINDING ACTIVITY AND COMPLEMENT (C1q, C3) CATABOLISM IN PRIMARY BILIARY CIRRHOSIS AND CHRONIC ACTIVE LIVER DISEASE [Abstract]. (Eng.) Thomas, H. C.; Potter, B. J.; Elias, E.; De Villiers, D.; Sherlock, S. (Royal Free Hosp., Pond St., London NW3 2QG, England). *Gastroenterology* 73(5):1250; 1977.

1469 LEVAMISOLE IN THE TREATMENT OF HBs ANTIGEN POSITIVE CHRONIC ACTIVE LIVER DISEASE [Abstract]. (Eng.) Thomas, H. C.; Chadwick, G.; Jain, S.; Sherlock, S. (Royal Free Hosp., Pond St., London NW3 2QG, England). *Gastroenterology* 73(5):1250; 1977.

1470 SIGNIFICANCE OF ISOENZYMES IN THE DIFFERENTIAL DIAGNOSIS OF ACUTE AND CHRONIC HEPATITIS. (Ger.) Graubaum, H. J.; Schimmelpfennig, W.; Schneider, G.; Klimmt, G.; Wagenknecht, C. (Hessische Strasse 3/4, DDR-104 Berlin, E. Germany). *Z. Gesamte Inn. Med.* 32(3):40-42; 1977.

See also, 1260, 1367, 1378, 1401, 1403, 1421, 1422, 1424, 1425, 1427, 1428, 1432, 1442, 1444, 1457, 1482, 1486, 1487, 1492.

LIVER AND BILIARY TRACT

Alcoholic Liver Disease

1471 IDIOPATHIC HEMOCHROMATOSIS AND IRON OVERLOAD IN ALCOHOLIC LIVER DISEASE: DIFFERENTIATION BY HLA PHENOTYPE. (Eng.) Simon, M.; Bourel, M.; Genetet, B.; Fauchet, R.; Edan, G.; Brissot, P. (Hopital Pontchaillou, 35043 Rennes, France). *Gastroenterology* 73(4):655-658; 1977.

Histocompatibility antigens (HLA) A and B were determined in 38 patients with idiopathic hemochromatosis (IH) and 22 patients with iron overload accompanying alcoholic liver disease (AH). As previously described, HLA A3 and HLA B14 antigens appeared more frequently in the IH group (76.3% and 28.9%, resp.) than in 204 controls (27% and 3.4%). In the AH group, the frequency of A3 (22.7%) and B14 (13.6%) was not significantly different from that observed in controls. The frequency of A3 was significantly lower in the AH group than in the IH group ($p<0.0001$). On this basis, a clear difference appears between the two conditions with iron overload, and genetic analysis of the results ruled out the hypothesis that AH would be a heterozygous form of IH exposed by alcohol.

1472 EARLY PERIVENULAR SCLEROSIS IN ALCOHOLIC FATTY LIVER: AN INDEX OF PROGRESSIVE LIVER INJURY. (Eng.) Van Waes, L.; Lieber, C. S. (Bronx Veterans Admin. Hosp., 130 West Kingsbridge Rd., Bronx, NY 10468). *Gastroenterology* 73(4):646-650; 1977.

To determine whether perivenular sclerosis, found in heavy drinkers with fatty liver, could be a precursor lesion of cirrhosis, controlled studies were performed in animal models and in humans. In alcohol-fed baboons that developed fibrosis or cirrhosis, progressive perivenular sclerosis invariably started at the fatty liver stage before or, even more commonly, in the absence of alcoholic hepatitis. No sclerosis occurred in controls or in alcohol-fed baboons and rats that did not progress beyond the fatty liver stage. Thirty of 75 patients who were heavy drinkers and had fatty liver (without other alcohol-induced liver changes) had a rim of fibrous tissue around the terminal hepatic venules that stained for collagen. None of 20 age-matched, nonalcoholic controls had

sclerosis. In five controls who consumed moderate amounts of alcohol (36-43% of total caloric intake) for up to 4 weeks, fatty liver developed, but perivenular sclerosis did not. The clinical and experimental data indicate that sclerosis around the terminal venules, a common but often overlooked complication of alcoholic fatty liver, reflects heavy prolonged drinking, and may identify those patients who are susceptible to develop the more advanced lesions of alcoholic liver injury upon continued drinking.

1473 TESTICULAR AND SPERMATOZOAL AUTOANTIBODY IN CHRONIC ALCOHOLIC MALES WITH GONADAL FAILURE. (Eng.) Van Thiel, D. H.; Gavaler, J. S.; Smith, W. I.; Rabin, B. S. (Univ. Pittsburgh Sch. Medicine, Pittsburgh, PA 15261). *Clin. Immunol. Immunopathol.* 8(2):311-317; 1977.

To determine whether immunity plays a part in the development or perpetuation of alcoholic gonadal injury, sera from 40 chronic alcoholic men, whose liver histology was classified by biopsy, were examined for the presence of autoantibodies utilizing a panel of tissue antigens. The frequency of autoantibodies detected in the alcoholic men was compared with that observed in 450 control men. Antibody only to spermatozoa and testicular antigens was detected in the alcoholic men at an increased frequency ($p \leq 0.001$). The reproductive function of the testes was evaluated in the alcoholic men. Of 22 alcoholic men requested to collect an ejaculate for evaluation, only 5 were potent enough to obtain a specimen for analysis. Two of these five alcoholic men were azoospermic and one was consistently oligospermic. All nine alcoholic men subjected to testicular biopsy demonstrated seminiferous tubular atrophy. Autoimmunity may play a role in the development or perpetuation of the observed gonadal failure present in alcoholic men.

1474 LIPOLYSOSOMES IN HUMAN LIVER: DISTRIBUTION IN LIVERS WITH FATTY INFILTRATION. (Eng.) Hayashi, H.; Winship, D. H.; Sternlieb, I. (Univ. Missouri Medical Center, Columbia, MO 65201). *Gastroenterology* 73(4):651-654; 1977.

The hepatocytes of 14 randomly selected patients with liver diseases were examined for the presence of lipolysosomes (lysosomes containing large lipid droplets). Organelles that displayed the same morphological characteristics and enzymatic activity as those originally described in the hamster and those found in the hepatocytes of patients with Wolman's or Wilson's disease were also found in hepatocytes of 13 of the 14 biopsy specimens that showed minimal to severe fatty infiltration. The numbers and sizes of the lipolysosomes seemed to increase with the degree of fatty infiltration, while their proportion in relation to the total number of lipid droplets remained below 5%. Lysosomes probably play an important role in hepatic lipid metabolism, and various pathogenetic mechanisms may trigger their proliferation.

1475 SERUM 25-HYDROXYVITAMIN D_3 LEVELS IN PATIENTS WITH LIVER DISEASE. (Eng.) Sonnenberg, A.; von Lilienfeld-Toal, H.; Sonnenberg, G. E.; Rohner, H. G.; Strohmeyer, G. (Stadtspital Triemli, Birmensdorferstr. 497, CH-8063 Zurich, Switzerland). *Acta Hepatogastroenterol.* 24(4): 256-258; 1977.

Serum 25-hydroxyvitamin D_3 levels were determined in 45 patients with chronic liver diseases by a radioreceptor assay and correlated with the serum albumin, calcium, and inorganic phosphate levels. The serum calcium level in the patients (4.4 ± 0.4 mEq/l) was significantly lower than that in 50 healthy controls (4.8 ± 0.2 mEq/l, $p<0.001$). The inorganic phosphate level in the patients (1.6 ± 0.3 mEq/l) was significantly lower than that in the controls (2.2 ± 0.4 mEq/l, $p<0.001$), but there was no significant correlation between the calcium level and the albumin or transaminase levels. The results indicate that 25-hydroxylation occurs mainly in the liver and that vitamin D absorption is impaired following liver injury.

1476 HEPATIC XENON-133 RETENTION: A NON-INVASIVE DIAGNOSTIC TEST FOR STEATOSIS [Abstract]. (Eng.) Ahmad, M. L.; Perrillo, R. P.; Sunwoo, Y. L.; Donati, R. M. (St. Louis Veterans Admin. Hosp., St. Louis, MO). *Gastroenterology* 73(5):1211; 1977.

1477 ENDOTOXIN AND BACTERIA IN PORTAL BLOOD [Letter to Editor]. (Eng.) Ansell, J.; Widrich, W.; Johnson, W.; Fine, J. (Veterans Admin. Hosp., Boston, MA 02130). *Gastroenterology* 73(5): 1190; 1977.

1478 THE COMPARATIVE SURVIVALS OF ALCOHOLICS VERSUS NONALCOHOLICS AFTER DISTAL SPLENORENAL SHUNT [Abstract]. (Eng.) Levi, J. U.; Zeppa, R.; Hutson, D. G.; Schiff, E.; Livingstone, A.; Fink, P. (Univ. Miami Sch. Medicine, Miami, FL 33152). *Gastroenterology* 73(5):1231; 1977.

1479 SERUM TRIIODOTHYRONINE (T_3) AS AN INDEX OF SEVERITY AND PROGNOSIS IN ALCOHOLIC LIVER DISEASE (ALD). EFFECT OF PROPYLTHIOURACIL (PTU) [Abstract]. (Eng.) Kalant, H.; Orrego, H.; Israel, Y.; Walfish, P. G.; Rankin, J.; Findlay, J. (Addiction Res. Foundation of Ontario, Toronto, Ontario, Canada). *Gastroenterology* 73(5):1227; 1977.

1480 AUTOIMMUNITY IN ALCOHOLIC HEPATITIS [Abstract]. (Eng.) Kanagasundaram, N.; Chen, T.; Leevy, C. M. (New Jersey Medical Sch., Newark, NJ). *Gastroenterology* 73(5):1227; 1977.

1481 LORAZEPAM ELIMINATION IN LIVER DISEASE [Abstract]. (Eng.) Kraus, J. W.; Marshall, J. P.; Johnson, R.; Wilkinson, G. R.;

Schenker, S. (Vanderbilt Univ. Sch. Medicine, Nashville, TN). *Gastroenterology* 73(5):1228; 1977.

1482 ELEVATION OF PLASMA ARGININE VASOPRESSIN LEVELS IN LIVER DISEASE [Abstract]. (Eng.) Hepner, G. W.; Weitzman, R. E. (Harbor General Hosp., Torrance, CA). *Gastroenterology* 73(5):1224; 1977.

1483 COMPARISON OF FEATURES OF ALCOHOLIC LIVER DISEASE [Abstract]. (Eng.) Connell, A. M.; Weir, D.; Fitzgerald, O.; McDonald, W.; Whelton, M.; McCarthy, C. M.; *et al.* (Univ. Hosp., Cincinnati, OH). *Gastroenterology* 73(5):1216; 1977.

1484 FREE WATER CLEARANCE (CH_2O) AND SODIUM BALANCE (SB) BEFORE AND AFTER PERITONEO-JUGULAR (PJS) AND SIDE-TO-SIDE PORTACAVAL SHUNTS (SSPS) [Abstract]. (Eng.) Capone, R. R.; Sarfeh, I. J.; Wong, P. Y. (Albany Medical Coll., Albany, NY 12208). *Gastroenterology* 73(5):1215; 1977.

1485 HIGH-DOSE SPIRONOLACTONE AS THE SOLE DIURETIC IN PATIENTS WITH REFRACTORY ASCITES [Abstract]. (Eng.) Campra, J.; Reynolds, T. B. (Univ. Southern California Sch. Medicine, Los Angeles, CA). *Gastroenterology* 73(5):1215; 1977.

See also, 0958, 1106, 1112, 1365, 1421, 1491, 1492, 1500, 1674.

LIVER AND BILIARY TRACT

Cirrhosis

1486 THE LAPAROSCOPIC STAGING OF PRIMARY BILIARY CIRRHOSIS. (Eng.) Lindner, H.; Dammermann, R.; Kloppel, G. (German Red Cross Hosp., Hamburg, W. Germany). *Endoscopy* 9(2):68-73; 1977.

The laparoscopic photographs of 38 cases of primary biliary cirrhosis with high mitochondrial antibody titers were reviewed to see if these photographs could be used as the basis of a system of laparoscopic staging. By scrutinizing the photographs taken with the Lumina photolaparoscope and reviewing the histological findings, the liver disease was divided into four stages: stage 1, nonspecific reactive hepatitis and yellowish-red coloration of the liver; stage 2, accentuation of yellowish-red pattern, increased signs of mesenchymal activity with conspicuous arterial vascularization and lymphatics, and granulomas and splenomegaly; stage 3, liver surface assumes a granular appearance with accentuation of the hexagonal lobule structure, but lobular regeneration is absent (the first signs of portal hypertension are recognizable); stage 4, development of nodularity on the liver surface and of a deepening greenish-blue color in advanced cases. Portal hypertension and decompensation are obvious features of this disease. However, by laparoscopy alone it is not possible to distinguish pure primary biliary cirrhosis from its mixed forms with chronic active hepatitis. It is suggested that intravital macropathology as revealed by the laparoscope is a valuable supplement to liver biopsy in the diagnosis and staging of primary biliary cirrhosis.

1487 EPIDEMIOLOGY OF CHRONIC LIVER DISEASES. (Eng.) Jorke, D.; Reinhardt, M. (Stadtisches Krankenhaus Jena, Dornburger Str. 159, DDR-69 Jena, E. Germany). *Acta Hepatogastroenterol.* 24(4):220-225; 1977.

The frequency and distribution of chronic liver diseases were studied in the East German population. In East Germany, mortality from liver cirrhosis decreases, in contrast to most other countries, although alcohol consumption is continuously increasing. In Berlin and in the industrialized districts, mortality from cirrhosis is higher than in the rural areas. Within a period of 6 yr, the number of inpatients with chronic liver diseases has risen from 11,600 to 14,200. From the epidemiologic point of view, viral hepatitis is not the main source of liver cirrhosis. The total recovery rate for acute hepatitis has increased from 80% to 94% over a period of 8 to 10 yr; liver cirrhosis was found to have developed only in 0.3%. Seventy percent of cirrhotic patients never suffer from viral hepatitis. Seventy-three percent of 1.8 million blood donors that were subjected to SGPT and hepatitis B surface antigen screening tests showed histologic changes of the liver. Using these screening methods it has been possible to register 7% of the persons among these blood donors as having liver diseases. Early recognition and treatment of chronic liver diseases seem to result in a reduction of mortality from liver cirrhosis.

1488 TOTAL FATTY ACIDS OF HUMAN LIVER IN RELATION TO AGE, SEX AND PATHOLOGY. (Eng.) Gershbein, L. L.; Baburao, K.; Pedroso, A. F. (Northwest Inst. Medical Res., 5656 W. Addison St., Chicago, IL 60634). *Acta Hepatogastroenterol.* 24(4):250-255; 1977.

Fatty acid composition was determined in lipids extracted from 28 normal, 3 fatty, and 2 cirrhotic adult livers removed at necropsy as well as from 4 newborn and infant organs. The total fatty acids were processed, the methyl esters submitted to gas chromatographic analysis, and the acid differences ascertained relative to age, sex, and pathology. The levels of unsaturated acids ranged higher than those of the saturated homologs and were even greater in the fatty liver lipids. The latter showed an increase in 18:1 and depressions in 18:0, 22:0, and 24:0 as compared with the normal specimens and the same changes in 18:0 and 18:1 as compared with the cirrhotic lipids.

1489 STUDY OF HEPATIC PORPHYRINS IN PATIENTS WITH LIVER CIRRHOSIS. (Spa.) Enriquez De Salamanca, R.; Solis Herruzo, J. A.; Arnalich, F.; Pardo Rueda, J. (Instituto de Medicina Experimental del C.S.I.C., Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 47(6):805-808; 1976.

Among 2,286 liver biopsy samples observed under UV light, 52 showed the red fluorescence (F+) characteristic of porphyria cutanea tarda (PCT), yet only 38/52 patients had dermal lesions and clinical symptoms of PCT. The remaining 14 F+ patients had chronic hepatopathies. Levels of the hepatic porphyrins (uro-, hepta-, hexa-, penta-, copro-, and protoporphyrin) in liver samples from seven F+ PCT patients and ten F+ hepatopathy patients were assessed by electrophoresis. Results were compared with those obtained in 20 patients with normal livers, 15 cirrhotic patients (F-), 6 patients with hepatic tumors, and 6 with cholestasis due to biliary obstruction. Total porphyrin levels were higher in the tissues of all 17 F+ patients than in normal tissues: 387 ± 119 µg/100 g for F+ patients without PCT, 5,935 ± 3,748 µg/100 g for F+ PCT patients versus 84 ± 31 µg/100 g for patients with normal livers. Total porphyrin levels were 140 ± 54 µg/100 g in F-cirrhosis patients, 372 ± 85 µg/100 g in patients with primary liver tumors, and 247 ± 73 µg/100 g in cholestasis patients. There was a higher proportion of uro- and heptaporphyrin in PCT liver tissue than in F+ non-PCT tissue. In the tumor tissue and cirrhosis F- and cholestasis tissues, protoporphyrin, and coproporphyrin predominated. The proportion of the different porphyrins was similar in all F- tissues to the proportion of porphyrin in normal tissue, whereas the proportions in both F+ groups were different from that in normal tissue. The similarities among the accumulated porphyrins in PCT patients and F+ chronic hepatopathy patients suggest that the same metabolic error may play a role in both disorders.

1490 ELECTROPHORETIC ANALYSIS OF URINARY PORPHYRINS IN HEPATIC CIRRHOSIS. (Spa.) Solis Herruzo, J. A.; Enriquez de Salamanca, R.; Pardo Rueda, J.; Arnalich, F. (Instituto de Medicina Experimental del C.S.I.C., Madrid, Spain). *Rev. Esp. Enferm. Apar. Dig.* 47(6):813-816; 1976.

The urine of 25 cirrhotic patients was analyzed to determine the incidence of porphyria cutanea tarda (PCT) in patients without the dermatological symptoms of this disease and the results were compared with those from 50 patients with PCT and 30 normal controls. Seventeen of the 25 cirrhotic patients had a marked history of alcoholism, 2 were Australia antigen positive, and 6 others were lacking these etiological factors. The uro-, hepta-, hexa-, penta-, and coproporphyrin fractions in the urine were separated by electrophoresis. A Doss index of PCT >14/100 indicated a carrier of the metabolic error of PCT. Eight of the cirrhotic patients had a pathologic PCT index. Such an anomaly in the electrophoretic pattern of the urinary porphyrins was confirmed by fluorescence tests; hepatic tissue gave a red fluorescence and had elevated levels of uro- and heptaporphyrins (54.5 ± 18.9 µg/day and 18 ± 10 µg/day, resp., versus 9 ± 7 µg/day and trace amounts, resp., in normal controls). No correlation was evident between the degree of the hepatopathy and the urinary pattern of the porphyrins. The metabolism of the porphyrins may be genetically altered in many of the patients, and this alteration may manifest itself in many ways, one of which is hepatopathy.

1491 THE CONVERSION OF D-XYLOSE INTO D-THREITOL IN PATIENTS WITHOUT LIVER DISEASE AND IN PATIENTS WITH PORTAL LIVER CIRRHOSIS. (Eng.) Pitkanen, E. (Univ. Central Hosp., Unioninkatu 38, SF-00170 Helsinki 17, Finland). *Clin. Chim. Acta* 80(1):49-54; 1977.

The extent of D-threitol formation in the oral D-xylose tolerance test and the role of the liver in D-xylose metabolism were studied. An oral D-xylose tolerance test was carried out in 12 patients with portal liver cirrhosis, in 7 patients with active fatty liver disease, and in 29 subjects without liver diseases. D-xylose and D-threitol were measured by means of gas-liquid chromatography. Fifteen percent of the D-xylose dose excreted in urine within 5 hr was recovered as D-threitol. The proportion of D-threitol was greater when the collection was extended to 24 hr. The D-threitol excretion was markedly diminished in cirrhotic patients, suggesting that a substantial proportion of the D-xylose-D-threitol conversion occurs in the liver. No decrease was detected in patients with fatty liver disease. No significant change in D-xylose excretion was observed in liver cirrhosis or in fatty liver disease. D-threitol can be regarded as the main end product of D-xylose metabolism in man.

1492 DEMONSTRATION OF ANTIBODIES AGAINST ALBUMINS IN CHRONIC HEPATOPATHIES: THEIR RELATION TO ALBUMIN TURBIDITY AND SOME OTHER TESTS. (Cze.) Fassati, P.; Jezkova, Z.; Spizek, J. (Oddeleni klinicke biochemie, OUNZ, Sokolovska 304, 190 00 Prague 9, Czechoslovakia). *Cas. Lek. Cesk.* 116(19):584-587; 1977.

Antibodies against albumins and their relation with albumin turbidity and other tests were studied in 50 patients with chronic hepatopathies (33 pa-

tients with liver cirrhosis or chronic hepatitis with clinical signs of developing cirrhosis [group 1], and 17 patients with hepatic steatosis [group 2]). Antibodies to albumins were found in 15 patients in group 1 (in 8/19 patients with medium degree of hepatic dysfunction and in 7/14 patients with severe dysfunction) and in 2 patients in group 2. Statistically significant positive correlation was found between the frequency of anti-albumin antibodies and the Takata reaction (correlation factor 0.402), the thymol turbidity test (correlation factor 0.411), and the gamma-globulin electrophoresis test (correlation factor 0.352) in group 1. The correlations between the anti-albumin antibodies and the albumin turbidity test and albumin and globulin electrophoresis tests were at the limit of statistical significance. The SGOT level correlated with the anti-albumin antibodies at the limit of statistical significance in group 2, but there were no other correlations.

1493 IMMUNE RESPONSE TO ϕX 174 IN MAN. 5. PRIMARY AND SECONDARY ANTIBODY PRODUCTION IN PRIMARY BILIARY CIRRHOSIS. (Eng.) Thomas, H. C.; Holden, R.; Jones, J. V.; Peacock, D. B. (Western Infirmary, Glasgow, Scotland). *Gut* 17(11):844-848; 1976.

To study changes in the immune response induced by primary biliary cirrhosis (PBC), 12 patients with this disease were subjected to i.v. antigenic challenge with the bacteriophage ϕX 174, and the IgM and IgG components of the primary and secondary immune responses were measured. The patients were challenged with ϕX 174 (2.0×10^9 plaque-forming U) on day 0 and again between the 28th and 42nd day after the first challenge. The primary immune response to ϕX 174 in the PBC patients was within the normal range, the peak mean titer being 760 ± 308. However, the secondary response was significantly decreased ($p<0.01$). There was no correlation between the degree of depression of the peak response and the severity of the disease, as shown by the degree of cholestasis or hepatocellular necrosis. All patients showed a marked increase in the total serum IgM concentration, and a moderate increase in IgG. The ratio of serum IgM to IgG was above normal in all cases. During the secondary response there was a higher proportion of IgM antibody than in normal subjects. Sheep RBC (E) rosette forming cells were reduced in concentration in the PBC patients, but there was no correlation with either the clinical or histological grading of the disease. There was a positive correlation between the primary and secondary antibody titers and the thymus-derived (T) cell concentration in the peripheral blood ($r=0.73$, p 0.01; $r=0.84$, $p<0.001$). EAC rosette-forming cells were reduced in some patients, and there was a positive correlation between the concentration of these cells and the magnitude of both the primary ($r=0.66$, $p<0.05$) and secondary response ($r=0.89$, $p<0.001$). There was a poor correlation between phytohemagglutinin responsiveness and E rosetting cell concentrations. Depressed T cell function in PBC may be partially responsible for the proportionately lower IgG compared with IgM antibody response.

1494 HYPOXIA AND HYDROTHORACES IN A CASE OF LIVER CIRRHOSIS: CORRELATION OF PHYSIOLOGICAL, RADIOGRAPHIC, SCINTIGRAPHIC, AND PATHOLOGICAL FINDINGS. (Eng.) Stanley, N. N.; Williams, A. J.; Dewar, C. A.; Blendis, L. M.; Reid, L. (Middlesex Hosp., Mortimer St., London W1N 8AA, England). *Thorax* 32(4):457-471; 1977.

The case of a 45-yr-old woman with liver cirrhosis complicated by cyanosis and recurrent right hydrothorax is described. A diagnostic pneumoperitoneum demonstrated that direct movement of ascites through a diaphragmatic defect was responsible for the hydrothoraces. Pulmonary function tests between episodes of hydrothorax showed severe arterial hypoxemia, a 23% right-to-left shunt, and a reduction in the carbon monoxide transfer factor to less than half the predicted value. Evidence of abnormal intrapulmonary arteriovenous communications was obtained by perfusion scanning. At necropsy the central tendon of the diaphragm showed numerous areas of thinning that were easily ruptured. Injection of the pulmonary arterial tree demonstrated precapillary arteriovenous anastomoses and pleural spider nevi. Morphometry provided quantitative evidence of pulmonary vasodilatation limited to the intra-acinar arteries, consistent with the effect of a circulating vasodilator. The scintigraphic and pathological findings suggested that shunting had been greater in the right than the left lung. Light microscopy of thin lung sections showed that the walls of small veins were thickened, and electron microscopy showed that this was due to a layer of collagen. The walls of capillaries were similarly thickened, which caused an approximately twofold increase in the minimum blood-gas distance and contributed to the reduction in transfer factor.

1495 EXERCISE-AGGRAVATED HYPOXEMIA AND ORTHODEOXIA IN CIRRHOSIS. (Eng.) Kennedy, T. C.; Knudson, R. J. (Univ. Arizona Coll. Medicine, Tucson, AZ 85724). *Chest* 72(3):305-309; 1977.

The case of a patient with a syndrome characterized by hypoxemia aggravated by exercise, orthodeoxia, hypocapnia, and evidence of hyperdynamic circulation complicating alcoholic cirrhosis is presented. The patient, a 63-yr-old man, first presented with dyspnea on exertion 4 yr after he had undergone a side-to-side portacaval anastomosis for bleeding esophageal varices. In addition to his other symptoms, there was hazy nodularity on chest x-ray films, clubbing, and reduced carbon monoxide diffusing capacity, but he had otherwise normal indices of pulmonary air flow, volume, and distribution of ventilation. The hypoxemia was more pronounced with the patient in the upright position and was further aggravated by exercise. This supports the presence of a shunt or shunt-like mechanism consisting of low-resistance vascular communications within the lung.

1496 TREATMENT OF REFRACTORY ASCITES BY REINFUSION OF THE ASCITIC FLUID. (Ita.) Sturniolo, G. C.; Farini, R.; Martin, A.; Chiara-

monte, M.; D'Angelo, A.; Ossi, E.; *et al.* (Cattedra Malattie Apparato Digerente, Istituto di Clinica medica I, Universita, Padova, Italy). *Recent. Prog. Med. (Roma)* 62(1):51-57; 1977.

Twenty-nine patients who developed ascites as a complication of cirrhosis were treated by reinfusion of concentrated ascitic fluid. Ascitic fluid was withdrawn from the abdominal cavity during peritoneal dialysis, concentrated by ultrafiltration, and reintroduced into the circulation through a vein in the arm. In the hour preceding introduction of the concentrated fluid, 1,000 ml of the unconcentrated ascitic fluid was recirculated through the body by a Watson Marlowe pump. Two hours after introduction of the concentrated fluid, patients were given 20-100 mg of furosemide i.v. Six hr after treatment began they were given a 3% solution of NaCl i.v. to compensate for the loss of electrolytes by ultrafiltration. Daily diuresis during the 5-day period preceding treatment and the 5-day period immediately after treatment showed a statistically significant increase of from 1.02 l to 1.5 l. There was no significant variation in azotemia, creatinemia, sodium or potassium levels, bilirubinemia or transaminase levels, and protein albumin or gamma globulin either before or after treatment. No major complications developed, although 48.8% of patients developed fever (not above 39 C), 53.3% developed chills, and 51% developed tachycardia (did not exceed 110 beats/min). Clinical improvement was observed for 2-27 months after treatment in 44% of the cases. Reinfusion of ascitic fluid is used for treatment of refractory cases and the majority of treated patients are in the final phases of the disease. It is difficult, therefore, to determine whether this treatment is effective solely in relieving symptoms or if it affects the ultimate course of the disease process.

1497 EFFECT OF β ADRENERGIC BLOCKING DRUGS IN THE RENIN-ALDOSTERONE SYSTEM, SODIUM EXCRETION, AND RENAL HEMODYNAMICS IN CIRRHOSIS WITH ASCITES. (Eng.) Wilkinson, S. P.; Bernardi, M.; Smith, I. K.; Jowett, T. P.; Slater, J. D. H.; Williams, R. (King's Coll. Hosp., Denmark Hill, London, SE5, England). *Gastroenterology* 73(4): 659-663; 1977.

The effects of propranolol and practolol on plasma renin activity were studied in 11 patients with cirrhosis and ascites. Their effects on the associated changes in aldosterone excretion, sodium excretion, and the intrarenal distribution of plasma flow were also investigated. As a result of effective β adrenergic blockade with either propranolol or practolol, plasma renin activity was suppressed in all 11 patients. In contrast, the effect on the rate of renal excretion of aldosterone was variable, suggesting that factors other than the renin-angiotensin system control the aldosterone secretion in cirrhosis. The changes in aldosterone could not be explained on the basis of changes in the plasma concentrations of potassium or sodium. The renal sodium excretion was inversely related to the values for aldosterone both before and after β adrenergic blockade, indicating a major role for aldosterone in regulating sodium excretion. A number of patients had an abnormal intrarenal distribution of plasma flow with a relative hypoperfusion of the renin-secreting outer cortical nephrons. Plasma renin activity was inversely related to outer cortical plasma flow, suggesting that the reduced outer cortical flow may be a stimulus to increased renin secretion. Because the abnormal intrarenal hemodynamic pattern was not corrected by suppression of plasma renin activity, and presumably angiotension II concentrations, it is unlikely that it is attributable to the known renal vasoconstrictor effects of angiotensin II.

1498 GROWTH HORMONE SECRETION AFTER L-DOPA TREATMENT OF LIVER CIRRHOSIS. (Ita.) Greco, A. V.; Ghirlanda, G.; Fedeli, G.; Rebuzzi, A.; Altomonte, L.; Manna, R. (Istituto di Patologia Medica, Universita Cattolica S. Cuore-Roma, Rome, Italy). *Minerva Med.* 67(47):3027-3032; 1976.

L-dopa (500 mg p.o.) was administered to 14 men with alcoholic cirrhosis of the liver and to 10 normal control subjects. L-dopa had no significant effect on plasma glucose or insulin levels in either the patients or controls. However, plasma levels of growth hormone (GH) were elevated in both groups. Ninety minutes after treatment, GH levels had risen from 1.97 ng/ml to 17.34 ng/ml in the controls and from 5.56 ng/ml to 28.03 ng/ml in the cirrhotics. These levels were significantly higher in the patients than in the controls. Three hours after treatment, GH levels returned to baseline values in both groups. This outcome was probably a reflection of the actual secretion of the hormone rather than its metabolism by the liver, since its rate of appearance and disappearance in the blood was the same in both the cirrhotics and controls.

1499 SURGICAL TREATMENT OF PORTAL HYPERTENSION BY MEANS OF SELECTIVE VASCULAR DERIVATIONS. (Spa.) Orozco, H.; Guevara, L.; Hernandez-Ortiz, J.; Elizondo, J.; Nieto, L.; Campuzano, M. (Natl. Inst. Nutrition, San Fernando Ave. and Tlalpan Viaduct, Mexico 22, D.F.). *Rev. Invest. Clin.* 29(2): 143-150; 1977.

Forty patients with portal hypertension were treated with selective venous shunts. Twenty patients had alcoholic cirrhosis, 16 had infectious cirrhosis, 1 had cirrhosis of unknown origin, and 3 had portal fibrosis. The 36 patients selected for the study who had elective operations were in a good state of general health otherwise, whereas the 4 patients who underwent emergency operation had profuse digestive hemorrhages and required immediate surgery. The surviving patients have been checked regularly every 3 months since the time of surgery. A selective end-to-end renosplenic shunt was performed in 24 patients (21 elective and 3 emergency operations); 9 patients had an elective distal splenorenal shunt (Warren), and 7 patients were treated by a selective end-to-side splenocaval shunt (4 elective without graft and 2 elective and 1 emergency with

Dacron grafts). Patient deaths within a month after surgery were considered as due to operative mortality. Mortality among the 36 patients who underwent elective surgery was 22%, and among the 4 who underwent emergency surgery it was 50%. Operative mortality for elective distal splenorenal shunt, selective end-to-end renosplenic shunt (elective), selective end-to-end renosplenic shunt (emergency), and selective end-to-side splenocaval shunt (elective) was 11.1%, 23%, 33%, and 33%, resp. The one patient who had emergency selective end-to-side splenocaval shunt died, bringing the total number of deaths to 10. Causes of death included infected ascites (1), liver failure (4), massive necrosis of the liver with reversal of portal venous blood flow (3), acute renal failure and hepatic failure (1), and acute cardiac, respiratory, and renal failure (1). Most frequent postoperative complications were upper gastrointestinal tract bleeding (7), hepatic insufficiency (24), and ascites (33). One patient had hepatic encephalopathy 2 yr after surgery. Survivors have no dietary restrictions, except for alcoholic beverages, and live normally.

1500 SURVIVAL IN PATIENTS WITH POSTNECROTIC CIRRHOSIS AND LAENNEC'S CIRRHOSIS UNDERGOING THERAPEUTIC PORTACAVAL SHUNT. (Eng.) Kanel, G. C.; Kaplan, M. M.; Zawacki, J. K.; Callow, A. D. (Univ. Chicago Hosp., 950 E. 59th St., Chicago, IL 60637). *Gastroenterology* 73(4, Part 1):679-683; 1977.

Survival rates were compared in 82 patients who underwent therapeutic portacaval shunt for Laennec's cirrhosis (45 cases), postnecrotic cirrhosis (29), or primary biliary cirrhosis (8). All patients were followed for at least 5 yr after shunt or until death. Survival rates were calculated by life-table methods. Eleven patients with postnecrotic cirrhosis had histological evidence of chronic active hepatitis. Survival rates were similar in alcoholic cirrhosis, postnecrotic cirrhosis, and primary biliary cirrhosis. Hepatic reserve provided the best criterion for predicting survival. The type of shunt, end-to-side, side-to-side, or splenorenal, did not influence survival. Histological evidence of chronic active hepatitis adversely affected survival in the patients with postnecrotic cirrhosis. However, histological evidence of ongoing alcoholic hepatitis in patients with Laennec's cirrhosis did not influence survival adversely. These data indicate that once a patient with cirrhosis has bled from esophageal varices, the etiology of the cirrhosis is not a major factor in determining survival after a therapeutic portacaval shunt.

1501 MESOCAVAL SHUNT IN PORTAL VEIN THROMBOSIS. (Ger.) Bergmann, H., Jr.; Brucke, P. (I. Chirurgische Abteilung, Allgemeines Krankenhaus der Stadt Linz, Krankenhausstrasse 9, A-4020 Linz, Austria). *Wien. Med. Wochenschr.* 127(12):364-368; 1977.

Two cases of mesocaval shunt operation in portal vein thrombosis are reported. Emergency mesocaval shunt operation was performed in a 48-yr-old man with portal hypertension and portal vein thrombosis who had undergone total gastrectomy. Mesocaval shunt was necessary for portal decompression due to massive parenchymal hemorrhage during splenectomy. Mesocaval shunt was performed routinely in a 10-yr-old boy with portal vein thrombosis and umbilical sepsis. The surgery was successful in both cases. Mesocaval shunt is indicated in portal vein thrombosis with ascites and esophageal varices and in children in whom splenorenal shunt is impossible due to the small diameter of the splenic and renal veins. It is also useful in all cases where splenorenal shunt is anatomically impossible, e.g., prehepatic block after extirpation of the spleen, thrombosed splenorenal shunt, intrahepatic block with cicatrization, and shrinkage or inflammation of the hepatoduodenal ligament.

1502 TRANSDUODENOPANCREATIC INTERPOSITION MESOCAVAL SHUNT USING AN INTERNAL JUGULAR VEIN AUTOGRAFT. (Eng.) Moreno Gonzalez, E.; Hebrero San Martin, J.; Solis Herruzo, J. A.; Rico Selas, P.; Rodriguez Agullo, J. L. (Digestive Surgery Dept., Ciudad Sanitaria de la Seguridad Social, Madrid, Spain). *Surg. Gynecol. Obstet.* 145(4): 565-569; 1977.

The use of transduodenopancreatic interposition mesocaval shunt using an internal jugular vein autograft is reported in 26 patients with bleeding esophageal varices, and the modification of this procedure in 4 patients with superior mesenteric vein anatomic abnormalities is described. These abnormalities made the usual subduodenal position of the jugular vein graft impossible. In these patients, a small pancreatectomy was performed between the transverse portion of the duodenum and the right aspect of the superior mesenteric vein, placing the interposition mesocaval shunt in a transduodenopancreatic position. No patient has had any bleeding after the operation, nor have esophageal varices been found at the time of the last examination (24 months after surgery). During this period, all patients showed a remarkable tolerance to proteins, and no patient has been on a low protein diet.

1503 HISTOCHEMICAL ACTIVITY OF γ-GLUTAMYLTRANSPEPTIDASE IN NORMAL AND CHOLESTATIC LIVER: AN INVERSE CORRELATION WITH INTRAHEPATIC ACCUMULATION OF BILE SALTS [Abstract]? (Eng.) Busachi, C. A.; Mebis, J.; Desmet, V. J. (Departement Medische Navorsing, Academisch Ziekenhuis Sint Rafael, Leuven, Belgium). *Digestion* 16(4):341; 1977.

1504 INCREASED TRANSCAPILLARY ESCAPE RATE OF ALBUMIN IN CIRRHOSIS AND ITS RELATION TO ASCITES FORMATION [Abstract]. (Eng.) Henriksen, J. H.; Parving, H. H.; Ranek, L.; Winkler, K.; Lassen, N. A. (Hvidovre Hosp., Copenhagen, Denmark). *Digestion* 16(4):347; 1977.

1505 FACTORS INFLUENCING ASCITES REABSORPTION. RELATIONSHIP TO DIURETIC-INDUCED AZOTAEMIA [Abstract]. (Eng.) Bosch, J.; Arroyo, V.; Sanz, G.; Estrada, L.; Rodes, J. (Hospital Clinico y Provincial, Barcelona, Spain). *Digestion* 16(4): 327; 1977.

1506 PARENTERAL 1,25-DIHYDROXYCHOLECALCIFEROL IN HEPATIC OSTEOMALACIA [Abstract]. (Eng.) Long, R. G.; Varghese, Z.; Meinhars, E.; Skinner, R. K.; Wills, M. R.; Sherlock, S. (Royal Free Hosp., London, England). *Digestion* 16(4):326; 1977.

1507 RENIN SECRETION RATE AND HEPATIC METABOLISM OF RENIN AND ALDOSTERONE IN CIRRHOSIS OF THE LIVER [Abstract]. (Eng.) Bosch, J.; Arroyo, V.; Mauri, M.; Rivera, F.; Navarro, F.; Rodes, J. (Hospital Clinico y Provincial, Barcelona, Spain). *Digestion* 16(4):349-350; 1977.

1508 ENZYME INDUCTION IN CIRRHOTIC PATIENTS DURING SPIRONOLACTONE THERAPY [Abstract]. (Eng.) Miguet, J. P.; Thebaut, A.; Dhumeaux, D. (Hopital Henri Mondor, Creteil, France). *Digestion* 16(4):353-354; 1977.

1509 USE OF β-ADRENERGIC BLOCKADE IN DIURETIC-INDUCED RENAL IMPAIRMENT IN CIRRHOSIS WITH ASCITES [Abstract]. (Eng.) Wilkinson, S. P.; Bernardi, M.; Wheeler, P. G.; Smith, I. K.; Williams, R. (King's Coll. Hosp., Medical Sch., London, England). *Digestion* 16(4):351; 1977.

1510 RADIOGRAPHIC VISUALIZATION OF OUTFLOW TUBE OF LEVEEN SHUNT FOR ASCITES: AN AID IN ASSESSMENT OF POSITION AND FUNCTION [Abstract]. (Eng.) Stanley, M. M.; Supan, W. A. P.; Greenlee, H. B.; Reinhardt, G. F. (Veterans Admin. Hosp., Hines, IL). *Gastroenterology* 73(5):1248; 1977.

1511 BONE DENSITOMETRY AND HISTOLOGY IN NON BILIARY CIRRHOSIS [Abstract]. (Eng.) Van Devanter, G.; Spjut, H.; Snyder, N. (Univ. Texas Medical Branch, Galveston, TX). *Gastroenterology* 73(5):1257; 1977.

1512 PRIMARY BILIARY CIRRHOSIS: COMPLEMENT ACTIVATION AND CHARACTERIZATION OF UNIQUE CIRCULATING IMMUNE COMPLEXES [Abstract]. (Eng.) Wands, J. R.; Dienstag, J. L.; Bhan, A. K.; Feller, E. R.; Isselbacher, K. J. (Massachusetts General Hosp., Boston, MA). *Gastroenterology* 73(5): 1253; 1977.

1513 BILE ACID METABOLISM IN PATIENTS WITH STABLE CIRRHOSIS [Abstract]. (Eng.) von Bergmann, K.; Mok, H. Y. I.; Grundy, S. M. (Veterans Admin. Hosp., La Jolla, CA). *Gastroenterology* 73(5):1253; 1977.

1514 OBSERVATION ON THE POSSIBLE ABSENCE OF A RELATIONSHIP BETWEEN LIVER CELL DAMAGE AND FIBROSIS [Abstract]. (Eng.) Tanyol, H.; (Chestnut Hill Hosp., Philadelphia, PA). *Gastroenterology* 73(5):1249; 1977.

1515 IMPORTANCE OF SCINTIGRAPHIC RESULTS FOR DETERMINING THE PROGNOSIS IN LIVER CIRRHOSIS. (Ger.) Loginov, A. S.; Petrova, O. P. (Zentralen Wissenschaftlichen Forschungsinstitut fur Gastroenterologie der Hauptverwaltung des Gesundheitswesens des Moskauer Exekutivkomitees, Moscow, USSR). *Radiol. Diagn. (Berl.)* 18(2):219-224; 1977.

1516 HEMORRHAGE IN PORTAL HYPERTENSION. (Ger.) Eckert, P.; Kortmann, K. B. (Chirurgische Klinik der Universitat, Martinistr. 2-52, D-2000 Hamburg 20, W. Germany). *Munch. Med. Wochenschr.* 119(37):1175-1178; 1977.

1517 CHOICE OF SURGERY FOR PORTAL HYPERTENSION. (Rus.) Uglov, F. G.; Zubtsovskii, V. N. (I. P. Pavlov First Leningrad Medical Inst., Leningrad, USSR). *Khirurgiia (Mosk.)* (9):3-6; 1977.

1518 SURGICAL TREATMENT OF LIVER CIRRHOSIS COMPLICATED BY ASCITES. (Rus.) Gulman, M. I.; Ershov, Yu. A. (All-Union Scientific Res. Inst. Clinical and Experimental Surgery, Moscow, USSR). *Khirurgiia (Mosk.)* (7):14-20; 1977.

1519 ANGIOGRAPHIC ROENTGENOSEMIOTICS OF LIVER CIRRHOSIS IN CHILDREN. (Rus.) Shumsky, V. I. (M. F. Vladimirskii Moscow Oblast Scientific Res. Inst. Clinical Medicine, Moscow, USSR). *Vestn. Rentgenol. Radiol.* (2):44-52; 1977.

1520 PRACTICAL DIAGNOSTIC PROBLEMS RAISED BY COMPLICATIONS IN THE COURSE OF CIRRHOSIS. (Fre.) Buffet, C.; Labayle, D.; Etienne, J. P. (Hopital Antoine-Beclere, 157, rue de la Porte-de-Trivaux, F 92141 Clamart, France). *Rev. Prat. (Paris)* 26(51):3601-3614; 1976.

1521 CLINICO-MORPHOLOGICAL TRAITS AND SOME ASPECTS OF LIVER CIRRHOSIS PATHOGENESIS. (Rus.) Krutskikh, E. V. (Leningrad Inst. Medicine and Hygiene, Leningrad, USSR). *Ter. Arkh.* 49(5): 113-116; 1977.

1522 LYMPHATIC VESSELS IN CIRRHOTIC LIVER. (Pol.) Zajac, S. (Z. Kliniki Chorob Naczyn i Transplantologii Instytutu Chirurgii AM w Warsawie i z Centrum Medycyny Doswiadczalnej i

Klinicznej PAN Kierownik Kliniki, Warsaw, Poland). *Pol. Przegl. Chir.* 49(3):329-333; 1977.

1523 DISORDERS OF HEMOSTASIS AND COAGULATION IN CIRRHOSIS. (Fre.) Larrieu, M. J. (Hopital de Bicetre, 94270 Bicetre, France). *Rev. Prat. (Paris)* 26(51):3651-3662, *passim*; 1976.

1524 TREATMENT OF CIRRHOTIC ASCITES. (Fre.) Levy, V. G. (Hopital Saint-Antoine, 184, rue du Faubourg-Saint-Antoine, 75012 Paris, France). *Rev. Prat. (Paris)* 26(51):3627-3637; 1976.

1525 SEA-BLUE HISTIOCYTE SYNDROME IN THAI SIBLINGS. (Eng.) Chainuvati, T.; Piankijagum, A.; Viranuvatti, V.; Silverstein, M. N. (Siriraj Hosp., Bangkok 7, Thailand). *Acta Haematol.* 58(1):58-64; 1977.

1526 HYPERNATREMIA AFTER THE ADMINISTRATION OF LACTULOSE [Letter to Editor]. (Eng.) Kaupke, C.; Sprague, T.; Gitnick, G. L. (Center Health Sciences, Univ. California at Los Angeles, 10833 Le Conte Ave., Los Angeles, CA 90024). *Ann. Int. Med.* 86(6):745; 1977.

1527 C6: SYNTHESIS BY THE LIVER IN VIVO. (Eng.) Hobart, M. J.; Lachmann, P. J.; Calne, R. Y. (Medical Sch., Hills Rd., Cambridge, England). *J. Exp. Med.* 146(2):629-630; 1977.

See also, 0895, 1102, 1106, 1118, 1136, 1227, 1260, 1323, 1365, 1375, 1378, 1384, 1393, 1401, 1403, 1421, 1428, 1459, 1463, 1464, 1475, 1477.

LIVER AND BILIARY TRACT

Gallbladder and Biliary Tract Diseases

1528 GALLBLADDER DISEASE--II. UTILIZATION OF THE LIFE TABLE METHOD IN OBTAINING CLINICALLY USEFUL INFORMATION: A STUDY OF 62,739 WEIGHT-CONSCIOUS WOMEN. (Eng.) Bernstein, R. A.; Giefer, E. E.; Vieira, J. J.; Werner, L. H.; Rimm, A. A. (Medical Coll. Wisconsin, 1725 W. Wisconsin Ave., Milwaukee, WI 53233). *J. Chronic Dis.* 30(8):529-541; 1977.

In a cross-sectional study of 62,739 women who were members of a club concerned with weight control problems, cohorts were reconstructed in a study to determine incidence rates and relative risks of gallbladder disease (GBD). The life-table technique was used to compare age-specific subgroups of the population by obesity levels and parity. This cohort analysis allowed the calculation of incidence rates which were used to evaluate risk factors of GBD. Relative risk and probability of GBD were also determined, and their application to a hypothetical patient was demonstrated. The probability of GBD significantly increased with increasing weight, age, and parity, in that order of importance. A multiple regression analysis revealed that obesity accounted for a large portion of the variation of GBD. About 88% of the variation was explained by the linear combinations of obesity, age, and parity. The risk of GBD among extremely obese women in their 20's was greater than six times that of women with normal weight in their 20's. For the entire population under study, the probability of developing GBD by age 60 was estimated to be 32.9/100 women.

1529 ACUTE CHOLECYSTITIS: 137 PATIENTS STUDIED BY INFUSION TOMOGRAPHY OF THE GALLBLADDER. (Eng.) Moncada, R.; Cardoso, M.; Danley, R.; Rodriguez, J.; Kimura, K.; Pickleman, J.; *et al.* (Loyola Univ. Medical Center, Maywood, IL 60153). *Am. J. Roentgenol.* 129(4):583-585; 1977.

Clinical experience with inflammatory cholecystopathy recognized in 137 patients studied by gallbladder infusion tomography is presented. The diagnostic accuracy of acute cholecystitis in 70 patients in whom this condition was suspected was 96% in positive and 94% in negative histologically proven cases. Infusion tomography of the gallbladder is an accurate, rapid, and safe diagnostic aid in the evaluation of acute cholecystitis. In cases of chronic cholecystitis, infusion tomography may be confirmatory but should not be relied on as a final diagnostic test due to a high percentage of false negative cases; only 64% of 18 cases of histologically proven chronic cholecystitis were diagnosed by this method.

1530 ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY (ERCP) PERFORMED IN CHILDREN. (Eng.) Urakami, Y.; Seki, H.; Kishi, S. (Sch. Medicine, Tokushima Univ., Kuramotocho, Tokushima City, 770, Japan). *Endoscopy* 9(2):86-91; 1977.

Endoscopic retrograde cholangiopancreatography (ERCP) was performed in three children to establish its

diagnostic value in young patients. A normal duodenofiberscope (JF-B2) was used with two patients (a 6-yr-old boy and 8-yr-old girl with colic abdominal pains) under general anesthesia and with a 13-yr-old boy (presenting with epigastric pain and abdominal discomfort) after premedication (1 ml Buscopan, i.m., and local anesthesia with xylocain spray). The technique of examination was the same as in adults. ERCP confirmed dilatation of the left hepatic and common bile ducts in the 6-yr-old boy and a cystic stricture of the extrahepatic bile duct and dilatation of the intrahepatic biliary tree in the 8-yr-old girl. ERCP also showed that the distal common bile duct was narrowed in both patients (extirpation of the cyst and hepatojejunostomy were performed). ERCP revealed numerous areas of narrowing and dilatation diffusely scattered throughout the pancreatic duct of the 13-yr-old boy. The common bile duct was partially visualized. Operation and autopsy disclosed a tumorous lesion involving the duct system (Burkitt's lymphoma). ERCP is recommended in every suspected case of choledochal cyst because it is superior to all other procedures in clarifying exactly the extention of the dilatation, the state of the distal common bile duct, and the relation between biliary and pancreatic duct systems; therefore an exact preoperative diagnosis can be made. Improvements in the duodenofiberscope for children may open the new field of ERCP also in newborns for the diagnosis and differentiation of congenital atresia of the common bile duct.

1531 PERCUTANEOUS CHOLANGIOGRAPHY WITH THE OKUDA NEEDLE. (Eng.) Hinde, G. De B.; Smith, P. M.; Craven, J. L. (Llandough Hosp., Nr. Penarth, South Glamorgan, Wales). *Gut* 18(8):610-614; 1977.

The results of percutaneous cholangiography with the Okuda needle in 42 consecutive patients with a clinical diagnosis of obstructive jaundice are presented. The steel needle used was smaller in diameter than the original Okuda needle (0.5 mm, 25 gauge versus 0.7 mm, 27 gauge). An aimed anatomical approach was made from the right flank to the area above the junction of the right and left hepatic ducts; this approach was achieved with the help of two lead markers. Six patients had intrahepatic cholestasis. The technique demonstrated the biliary anatomy in 41 patients, and the radiological diagnosis was confirmed by laparotomy, necropsy, or liver biopsy. There were no significant complications. Laparotomy, when indicated, was performed earlier in the course of the jaundice, and it was avoided in seven patients. Precise knowledge of the site of the obstruction was of great help to the surgeon. This technique represents an important advance in the management of the jaundiced patient. The rarity of complications is attributed to prophylactic antibiotics, the slimness and flexibility of the needle, and the lateral entry point, which provides protection against bile leakage through the thickness of the liver.

1532 PLASMA DISAPPEARANCE AND CONJUGATION OF [^{14}C]CHOLIC ACID IN PATIENTS WITH OBSTRUCTIVE JAUNDICE. (Eng.) Horak, W.; Knoflach, P.; Schuster, E.; Kemmenesi, W.; Grabner, G. *In: Bile Acid Metabolism in Health and Disease. Proceedings of the IV Bile Acid Meeting held at the Hilton Hotel, Basel, Switzerland, October 11-13, 1976.* University Park Press. (Baltimore, MD): 247-251; 1977.

To investigate what mechanism (i.e., impaired uptake, conjugation, or excretion) is involved in the pathogenesis of impaired bile acid clearance in patients with obstructive jaundice, plasma disappearance and conjugation of i.v.-injected free [^{14}C]-cholic acid were investigated in five healthy subjects and in seven patients with obstructive jaundice. Radioactivity was measured in plasma samples and in the individual plasma bile acid fractions after separation by thin-layer chromatography. Plasma curves of free, conjugated, and total [^{14}C]radioactivity were calculated by an iterative nonlinear least-squares fitting procedure using a computer. A compartment model was applied to estimate the transfer rates of [^{14}C]cholic acid between free and conjugated plasma, liver, and bile compartments. In the patients, the disappearance of total plasma radioactivity was greatly prolonged, while the plasma disappearance of free [^{14}C]cholic acid was nearly normal. The impaired disappearance of total plasma radioactivity could be attributed to a slight decrease of hepatic uptake and to a considerable reflux of conjugated [^{14}C]cholate from liver to plasma. While the conjugation rate was similar to healthy subjects, the biliary excretion rate was practically null in obstructive jaundice. Free [^{14}C]cholic acid plasma clearance may allow an estimation of hepatic bile acid uptake and conjugation independently from disorders of biliary excretion, while large amounts of conjugated [^{14}C]cholate appearing in plasma indicate cholestasis. This test therefore may provide new information for the differential diagnosis of the patient with jaundice.

1533 COMPOSITION OF BILE IN POPULATIONS AT ELEVATED RISK OF CHOLELITHIASIS. (Ita.) Ponz de Leon, M.; Ferenderes, R.; Carulli, N. (Istituto di Clinica Medica Generale e Terapia Medica della Universita, Modena, Italy). *Minerva Med.* 67 (53):3483-3490; 1976.

The cause of cholelithiasis in diabetics and cirrhotics was sought by studying the composition of bile in 14 controlled diabetics, 10 cirrhotics, 15 cholelithiasis patients, and 8 controls. Samples were taken after a fast of at least 12 hr using a double radiopaque catheter so that separate samples of gastric and duodenal juices could be obtained. Study of bile acids, phospholipids, and cholesterol showed a significantly elevated index of saturation in diabetics compared to controls ($p<0.0025$), with a lesser but still significant elevation in cholelithiasis patients ($p<0.01$) and no significant elevation in cirrhosis patients. Elevation of the index was related to a decrease in the level of bile acids and an increase in the level of cholesterol. In terms of total composition (expressed in percent), cholesterol in controls was 5.6 ±

2.0 compared to 9.5 ± 2.5 in diabetics (p=0.005), but 7.5 in cholelithiasis (not significant). In the diabetics and cirrhotics, phospholipids were increased in comparison to controls (18.5 ± 8.3 and 12.7 ± 7.0 versus 16.4 ± 7.9%), but not to a significant level. Bile acids in diabetics, cholelithiasis patients, and cirrhotics were decreased compared to controls, but again not to a significant degree. Thus, there are clear physiopathological changes in bile composition in diabetics to explain the occurrence of cholelithiasis, but no cause for cholelithiasis in cirrhotics was elucidated by these studies.

1534 CHANGES IN BILE LIPIDS ACCOMPANYING OOPHORECTOMY IN A PREMENOPAUSAL WOMAN. (Eng.) Bennion, L. J. (Natl. Inst. Health, 4212 N. 16th St., Phoenix, AZ 85016). *N. Engl. J. Med.* 297(13):709-711; 1977.

Changes in bile lipids were examined in a premenopausal woman (36 yr old) who underwent bilateral oophorectomy. Oophorectomy was carried out because of adenocarcinoma of the endometrium. Bile acid pool size, bile acid composition, and gallbladder bile lipid composition were studied 2 months before and again 3 months after surgery. Whereas there was no substantial change in the relative proportions of bile acids, the size of the total bile acid pool increased from 1,040 mg before to 2,170 mg after the operation. The gallbladder bile cholesterol concentration was decreased after oophorectomy (4.8 after versus 7.0 mM before). Cholesterol saturation was 100% before and 81% after surgery, according to one method and 70% before and 49% after, according to a second method. This change suggests that endogenous ovarian hormones may influence the propensity to gallstone formation in a manner similar to that of exogenous estrogens and progestins.

1535 EFFECT OF PROLONGED FEEDING WITH CHENODEOXYCHOLIC ACID ON BILE IN PATIENTS WITH AND WITHOUT GALLSTONES. (Eng.) Bateson, M. C.; Ross, P. E.; Murison, J.; Bouchier, I. A. D. (Ninewells Hosp. and Medical Sch., Dundee, Scotland). *Gut* 18(8):599-605; 1977.

The effect of prolonged chenodeoxycholic acid therapy on bile cholesterol saturation was studied. Nineteen patients who received chenodeoxycholic acid (750 mg/day) for 6 months had duodenal bile aspirated before and after treatment. In five patients with hypertriglyceridemia but no gallstones, cholesterol saturation was reversed in every case, the mean cholesterol saturation index (SI) changing from 1.38 ± 0.31 to 0.68 ± 0.06 ($p<0.005$). In 14 patients with gallstones, there was also an improvement in bile cholesterol content, but this change was not sufficient to produce mean unsaturation, the SI changing from 1.55 ± 0.52 to 1.13 ± 0.43 ($p<0.05$). Only 7 of 14 patients with gallstones achieved cholesterol unsaturation. In four patients with hypertriglyceridemia and gallstones, mean unsaturation was produced and the SI changed from 1.70 ± 0.45 to 0.86 ± 0.47 ($p<0.05$). When all nine patients with hypertriglyceridemia were grouped, the mean fell from 1.52 ± 0.40 to 0.76 ± 0.30 after therapy ($p<0.001$). In contrast, the 10 patients without hypertriglyceridemia showed no significant fall in SI, which was 1.50 ± 0.54 before and 1.24 ± 0.40 after therapy. The ability of chenodeoxycholic acid feeding to improve bile saturation with cholesterol correlated with the presence of hypertriglyceridemia whether or not gallstones were present. It did not correlate with gallstone dissolution or body weight.

1536 CHANGES OF BILIARY BILE ACIDS IN PATIENTS AFTER ADMINISTRATION OF URSODEOXYCHOLIC ACID. (Eng.) Makino, I.; Nakagawa, S. *In: Bile Acid Metabolism in Health and Disease. Proceedings of the IV Bile Acid Meeting held at the Hilton Hotel, Basel, Switzerland, October 11-12, 1976.* University Park Press (Baltimore, MD): 211-217; 1977.

The value of ursodeoxycholic acid (UDCA) for gallstone dissolution was studied in 44 patients with radiolucent gallsones. The patients were randomly allocated to one of three treatment regimens: placebo (lactose) or 150 or 600 mg/day of UDCA for 6 months. No patient in the placebo group responded. Unequivocal dissolution of gallstones was observed in 8 of 31 patients receiving UDCA; 38% of the patients receiving the lower dose and 13% of those receiving the higher dose responded. UDCA possessed neither apparent hepatotoxicity nor cathartic activity. Desaturation of biliary cholesterol was induced by UDCA administration, and UDCA in bile increased in proportion to dose of UDCA. The major fecal bile acid of patients receiving UDCA was lithocholate. Serum bile acid levels rose slightly after UDCA treatment (2.4 ± 1.7 µg/ml), and UDCA occupied over 50% of total bile acid. These results confirm the efficacy of UDCA for gallstone dissolution.

1537 STRUCTURAL ALTERATIONS OF THE LIVER IN CHOLELITHIASIS. (Ita.) Marenco, G.; Menardo, G.; Gazzaniga, G. M.; Conti, U. (Istituti Ospedalieri Santa Corona, Pietra Ligure, Italy). *Pathologica* 69(987/988):77-85; 1977.

Structural alterations in the liver were studied in 85 patients with calculi of the gallbladder (60 patients) or of both the gallbladder and the common bile duct (20 patients). Of the 60 patients with calculi of the gallbladder 20 were asymptomatic. Each patient had surgical biopsy of the liver at some distance from the cholelithiasis. Bile duct calculi cause inflammatory infiltration into the portal spaces, proliferation of the bile ducts at the margin of the portal spaces, inflammatory infiltration into the interlobular spaces, and biliary hardening. Gallbladder were characterized by chronic inflammation and fibrotic lesions of the portal spaces. Structural alterations of the liver were most pronounced in patients with a clinical history of frequent attacks of colic. These structural alterations occurred in about 85% of the cases

and were different than the morphologic pattern of chronic aggressive hepatitis.

1538 MORTALITY OF OPERATIONS ON GALLBLADDER AND BILE DUCTS PERFORMED IN A TRAINING HOSPITAL. (Eng.) Schothorst, A. (Dept. Surgery, St. Geertruiden Gast- of Ziekenhuis, Deventer, Netherlands). *Arch. Chir. Neerl.* 29(2):109-116; 1977.

A retrospective study was made of the mortality rate from 990 operations done during a 6-yr period (1967-1972) at the St. Geertruiden Gasthuis of Ziekenhuis to determine whether surgery in a training hospital adversely affected the mortality rate. Five groups of operations were studied: 1) cholecystectomy (397, of which 234 operations were by surgeon), 2) cholecystostomy (5), 3) cholecystectomy plus choledochotomy (504, of which 281 operations were by surgeon), 4) re-exploration or inspection of the biliary tract (44, all by a surgeon), and 5) operations necessitated by malignant neoplasia in the region of the gallbladder or bile ducts and pancreas (40, all by a surgeon). Zero percent surgical mortality was shown in assistant-operated patients, suggesting a low risk factor for patients with biliary tract pathology; the low mortality was attributed to preoperative selection of patients to be operated on by assistants or by surgeons. A higher incidence of complications and mortality is suggested in the group over 70 yr of age, but age was no criterion in determining whether an operation was to be done by an assistant. It is concluded that the training system does not work to the patient's disadvantage.

1539 ULTRASONIC SCANNING IN POSTCHOLECYSTECTOMY CHOLEDOCHOLITHIASIS. (Eng.) Sapala, M. A.; Steel, W. B.; Resto Soto, A. D.; Sapala, J. A. (Hutzel Hosp. and Wayne State Univ. Sch. Medicine, 4707 St. Antoine, Detroit, MI 48201). *Surgery* 84(4):420-424; 1977.

Four patients underwent ultrasonic scanning of the right upper quadrant for the evaluation of unexplained jaundice and fever. Each patient had undergone a previous cholecystectomy with common bile duct exploration for biliary tract calculi. No patient at that time revealed retained stones on either intraoperative or postoperative cholangiograms. In none of the patients studied was the exact nature of the obstruction appreciated by the clinical and laboratory data. All patients revealed biliary duct dilatation on ultrasound, and the majority demonstrated a bile duct calculus as well. The authors do not recommend that ultrasonic scanning be substituted for p.o. or i.v. cholangiography when the latter can be performed. Nor should ultrasonic scanning be used to replace endoscopic retrograde cholangiopancreatography, percutaneous transhepatic cholangiography, or even liver biopsy if these procedures are indicated and can be completed with a reasonable degree of safety. However, ultrasound can be very helpful in the occasional patient whose biliary tract obstruction defies exact diagnosis when routine dye studies and invasive diagnostic procedures are neither accurate nor feasible.

1540 TRANSAMPULLARY SEPTECTOMY FOR POST-CHOLECYSTECTOMY PAIN. (Eng.) Moody, F. G.; Berenson, M. M.; McCloskey, D. (Univ. Utah Coll. Medicine, Salt Lake City, UT 84132). *Ann. Surg.* 186(4):415-423; 1977.

A systematic prospective study of transampullary septectomy combined with sphincteroplasty was carried out in patients with severe, long-standing postcholecystectomy pain. Twenty-eight patients had excision of the common wall between the terminal bile duct and duct of Wirsung (ampullary septum). Twenty-two also had a sphincteroplasty; six had had this procedure previously. Pancreatic function studies, scintiscans, ultrasound, and pancreatograms were nondiagnostic. Hyperamylasemia was an uncommon finding. Eight patients had evidence of mild pancreatitis at exploration. There was gross scarring of the ampullary septum in 22 cases. Histologic examination revealed inflammation in 12 septa; the degree of fibrosis could not be assessed, since 14 control septa from autopsy material free from biliary tract disease revealed a comparable degree of collagen and smooth muscle. There were no deaths, and minimal morbidity. In follow-up from 7 to 59 months (mean=26), 16 patients are relatively free of pain, 5 have occasional episodes that require non-narcotic analgesics, and 7 have gained no relief from the operative procedure. A randomized controlled trial is recommended.

1541 INFANTILE HEPATITIS AND CONGENITAL ATRESIA OF BILE DUCTS. (Eng.) Watanabe, W.; Kobayashi, O. (Niigata Univ. Sch. Medicine, Niigata, Japan). *Acta Med. Biol.* 23(3/4):191-213; 1976.

Etiological, clinical, and histological data on 67 infants with infantile hepatitis and 41 infants with congenital biliary atresia observed from 1961 to 1974 are reported. Comparison of prenatal histories showed that the infants with hepatitis had shorter average gestation, lower birth weights, and familial incidence of disease. Association of other congenital anomalies such as Down's syndrome, microcephalus, and cerebral palsy were observed with Tests for Australia antigen were positive in 1/20 infants with hepatitis tested and negative in all 7 infants with biliary atresia tested. Histological examination of biopsy specimens can readily distinguish between the two diseases in the early stages. Ductal proliferation appears to be the most reliable sign of biliary malformation. The severe cases of infantile hepatitis are difficult to distinguish from biliary atresia. Electron microscopy was the method of choice for differentiating between the two syndromes. Infantile hepatitis and congenital atresia of the bile ducts may be analogous diseases caused by viral infections or noxious agents during the first trimester of pregnancy.

1542 "CYSTS" OF THE COMMON BILE DUCT (DYSPLASIA OF THE COMMON BILE DUCT). (Ita.) Pezzi, A.; Jasonni, V.; Brusori, G. (Policlinico S. Orsola, Bologna, Italy). *Minerva Med.* 67(7):443-450; 1976.

A review of the salient features of cholecystic dysplasia is given. Extrahepatic cholecystic dysplasia is classified into choledochal cysts, choledochal diverticuli, and choledoceles. Choledochal diverticuli cause torsion and strangulation and often result in stenosis, which extends to the superior margin of the pancreas. Choledoceles cause the ampulla of Vater to protrude into the duodenum; there is also some slight tumifaction at the extreme distal end of the biliary-pancreatic path. Congenital ectasia is a severe form of cholecystic dysplasia that causes extensive involvement of the liver and that may result in renal failure. Cases with systemic and intra- and extrahepatic involvement are classified as type IV cholecystic dysplasia and are characterized by involvement of the primary bile duct.

1543 APLASIA OF THE GALLBLADDER WITH COMMON DUCT STONES. (Eng.) Wahlby, L. (Univ. Hosp. Umea, Umea, Sweden). *Acta Chir. Scand.* 143(4):241-243; 1977.

The case of a 79-yr-old woman with gallbladder aplasia and common duct stones is reported. The patient presented with right upper quadrant tenderness and a 2-week history of jaundice. She did not respond to conservative treatment, and the serum bilirubin level increased to 205 μM. A preoperative cholangiography revealed no gallbladder or cystic duct. At laparotomy, the liver was enlarged, icteric, and slightly fibrotic. No gallbladder or cystic duct was present. The common duct was 25 mm wide and contained four faceted stones. The patient remains asymptomatic 4 yr after choledocholithotomy and drainage. Only 22 cases of aplasia of the gallbladder with commmon duct stones have previously been reported.

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1616 SPONTANEOUS HEMATOBILIA IN THE DIFFERENTIAL DIAGNOSIS OF UPPER GASTROINTESTINAL BLEEDING. (Ger.) Jaeger, M.; Williman, P. (Medizinische Klinik des Stadtspitals Waid, CH-8037, Zurich, Switzerland). *Schweiz. Med. Wochenschr.* 106(41): 1392-1395; 1976.

1617 MICROBIAL JAUNDICE. (Fre.) Savoye, B. (No affiliation given). *J. Med. Lyon* 57(1303):355-358; 1976.

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See also, 1090, 1095, 1097, 1099, 1101, 1102, 1106, 1108, 1117, 1131, 1132, 1134, 1227, 1231, 1232, 1234, 1260, 1270, 1315, 1323, 1350, 1375, 1377, 1384, 1391, 1397, 1477.

GENERAL

1619 RELATIVE IMPORTANCE OF VIRUSES AND BACTERIA IN THE ETIOLOGY OF PEDIATRIC DIARRHEA IN TAIWAN. (Eng.) Echeverria, P.; Ho, M. T.; Blacklow, N. R.; Quinnan, G.; Portnoy, B.; Olson, J. G.; Conklin, R.; *et al.* (Harvard Medical Sch., 25 Shattuck St., Boston, MA 02115). *J. Infect. Dis.* 136(3):383-390; 1977.

Eighty Taiwanese children with diarrhea were studied to determine the relative importance of (a) reovirus-like agent (RVLA), (b) *Escherichia coli* that produce heat-stable and heat-labile toxin, (c) invasive *E. coli*, (d) *Salmonella, Shigella,* and *Vibrio,* (e) enteroviruses, and (f) parasites in the etiology of pediatric diarrhea. Of 75 patients tested for evidence of infection with RVLA by electron microscopy of stools and/or by assays of complement-fixing and immunofluorescent antibody response to the serologically related Nebraska calf diarrhea virus, 42 were positive. Of children whose stools contained the agent, 90% showed a fourfold or greater rise in titer of immunofluorescent antibodies, and 85% developed a fourfold or greater rise in titer of complement-fixing antibodies to the agent. Strains of *E. coli* producing only heat-labile toxin (LT) were isolated from 7% of 57 ill children who had not received previous antibiotics and from 4% of 131 healthy control children (p=0.49). Strains of *E. coli* producing only heat-stable toxin (ST) were isolated from 5 of 57 age-matched children in the 131 healthy controls tested. Invasive *E. coli* were not found. In addition, three of six children with bacterial and/or serological evidence of an infection with LT-producing *E. coli* and four of five children infected with ST-producing *E. coli* had infections with RVLA. RVLA appeared to be the major cause of diarrheal illness in Taiwan during the summer of 1975, whereas toxigenic *E. coli* appeared to be responsible for a smaller proportion of pediatric enteritis.

1620 SYMPTOMATIC TREATMENT OF DIARRHEA WITH BISMUTH SUBSALICYLATE AMONG STUDENTS ATTENDING A MEXICAN UNIVERSITY. (Eng.) DuPont, H. L.; Sullivan, P.; Pickering, L. K.; Haynes, G.; Ackerman, P. B. (6400 West Cullen St., Houston, TX 77030). *Gastroenterology* 73(4, Part 1):715-718; 1977.

The value of bismuth subsalicylate in the symptomatic treatment of diarrhea was studied in students attending a Mexican university who developed diarrhea. One hundred eleven students were given 30 ml each half hour until eight doses (total dose of active drug, 4.2 g) were given, and 58 students received twice this dose (8.2 g of active drug) over the 3.5-hr treatment period. The number of unformed stools was significantly decreased in both bismuth subsalicylate treatment groups compared to the placebo controls for the period 4 to 24 hr after therapy. A reduction in diarrhea was addi-

tionally noted for the duration of the 48-hr surveillance period for the students receiving the higher dose of active drug. Subjective relief within 24 hr of therapy of the symptoms of diarrhea, nausea, and abdominal pain or cramps was reported with a significantly increased frequency in the bismuth subsalicylate group. The most pronounced effect of the treatment occurred in the United States students with diarrhea who had recently arrived in Mexico. This appeared to be related to the favorable effect of bismuth subsalicylate on the course of toxigenic *Escherichia coli* infection. The thirty-two students with shigellosis did not experience a prolonged illness in either treatment group. Bismuth subsalicylate is effective in reducing diarrhea, regardless of etiology.

1621 THORACIC SPINAL CORD TUMOR PRESENTING WITH DYSAUTONOMIC DIARRHEA. (Eng.) Fricke, R. D.; Romine, J. S. (Veterans Admin. Section, 3350 La Jolla Village Drive, San Diego, CA 92161). *Gastroenterology* 73(5):1152-1156; 1977.

The case of a 52-yr-old man with an ependymoma who presented with dysautonomic diarrhea is described. The patient had incapacitating diarrhea of 1-month duration and developed orthostatic hypotension and progressive long tract central nervous system signs. No enteric disease was found to account for the diarrhea. An intramedullary midthoracic spinal cord tumor was found. Diarrhea and hypotension resolved after local cord decompression and irradiation of the tumor. Dysmotility caused by interruption of thoracic sympathetic pathways to the gut was considered responsible for the diarrhea, and altered splanchnic hemodynamics for the hypotension. The regional autonomic neuroanatomy and the known effects of the autonomic nervous system upon bowel function and the splanchnic circulation explain the patient's symptoms.

1622 MASSIVE RECURRENT HEMORRHAGE OF THE DISTAL PART OF THE DIGESTIVE TRACT DUE TO HEREDITARY HEMORRHAGIC TELANGIECTASIA (OSLER'S SYNDROME). (Fre.) Besson, A.; Saegesser, F. (Service de Chirurgie A, CHUV, CH-1011 Lausanne, Switzerland). *Schweiz. Med. Wochenschr.* 107(30):1057-1064; 1977.

Hereditary hemorrhagic telangiectasia was diagnosed in a 67-yr-old woman with multiple angiomas of the head, trunk, and fingers by emergency selective angiography of the celiac trunk and superior and inferior mesenteric arteries during uncontrollable rectal hemorrhage. The investigations revealed a large arteriovenous fistula in the cecal region, originating from a large ruptured telangiectasia. Right hemicolectomy with anastomosis between the ileum and ascending colon under angiographic control of the resected specimen was successful; the patient was in good condition more than 1 yr after surgery. The patient's brother has the same disease, and their mother had frequent epistaxis, a typical sign of telangiectasia.

1623 REGULATION OF POOL SIZE OF BILE ACIDS IN MAN. (Eng.) Mok, H. Y. I.; von Bergmann, K.; Grundy, S. M. (Veterans Admin. Hosp., 3350 La Jolla Village Drive, San Diego, CA 92161). *Gastroenterology* 73(4, Part 1):684-690; 1977.

Various components of the enterohepatic circulation were investigated in 33 subjects to determine their effects on bile acid pool size. Seven patients had increases in plasma cholesterol with near normal triglycerides, and six others had raised plasma triglycerides. The pool size and secretion rates of bile acids were measured using an intestinal perfusion method, and bile acid synthesis was determined by fecal excretion. From these measurements, cycling frequency of the pool and intestinal absorption of bile acids were calculated. The results were analyzed for the group as a whole and for four subgroups based on incremental pool sizes (1-2 g, 2-3 g, 3-4 g, and >4 g). Factors that should regulate pool size are synthesis, absorption efficiency, and cycling frequency of the pool. In subjects with relatively low pools, steady state synthesis and absorption of bile acids were not reduced. However, two other factors appeared to be especially important in keeping pool sizes in the lower ranges--a high cycling frequency and overly sensitive feedback inhibition of bile acid synthesis. First, there was a significant inverse correlation between cycling frequency and pool size. Theoretically, a high cycling frequency should reduce pools in two ways--by suppressing synthesis and by increasing fecal losses. Second, in patients with low pools, transhepatic flux of bile acids was also reduced, and daily synthesis of bile acid, although being in the same range as those with larger pools, was inappropriate for the magnitude of the flux. With this overactive feedback on bile synthesis, these patients were unable to increase their bile acid synthesis to supranormal levels so as to cause a reexpansion of the pool to higher levels.

1624 Salmonella carmel INFECTION IN INFANCY: CLINICAL OBSERVATIONS. (Eng.) Berant, M.; Wagner, Y.; Cohen, N.; Kaufstein, M. (Hillel Yaffe Government Hosp., Hadera, Israel). *Am. J. Trop. Med. Hyg.* 26(5, Part 1):1048-1049; 1977.

1625 TYPHOID FEVER IN PREGNANCY: WITH PROBABLE TYPHOID HEPATITIS. (Eng.) Schiffman, P.; Samet, C. M.; Fox, L.; Neimand, K. M.; Rosenberg, S. T. (Harbor General Hosp., Torrance, CA 90503). *N.Y. State J. Med.* 77(11):1778-1779; 1977.

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(METAMUCIL) [Abstract]. (Eng.) Walan, A.; Bergdahl, B.; Skoog, M. L. (Univ. Hosp., Linkoping, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):111; 1977.

1628 INTRACTABLE DIARRHEA AND THE COLON [Letter to Editor]. (Eng.) Michel, M. V.; Martuscelli, A.; Lebenthal, E. (Hospital de Pediatria, Centro Medico Nacional, Mexico City, Mexico). *Pediatrics* 60(4):565-566; 1977.

1629 BLOOD FLOW DISTURBANCES IN THE GASTROINTESTINAL TRACT. (Ger.) Koch, H. (Med. Univ.-Klinik, Krankenhausstrasse 12, 852 Erlangen, W. Germany). *Fortschr. Med.* 95(17): 1155-1159; 1977.

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1631 CARRAGEENAN AND NECROTIZING ENTEROCOLITIS OF THE NEWBORN [Letter to Editor]. (Eng.) Udall, J. N.; Suskind, R. M.; McGill, H. C., Jr.; Sprinz, H. (Clinical Res. Center, Massachusetts Inst. Technology, Boston, MA 02142). *Gastroenterology* 74(1):161-162; 1978.

1632 THE POSTOPERATIVE DERANGEMENTS OF FAT ABSORPTION FOLLOWING G-I TRACT SURGERY--A NEW ABSORPTION TEST WITH PURIFIED ^{131}I-TRIOLEIN [Abstract]. (Eng.) Koyama, S.; Hatakeyama, K.; Yamashita, Y.; Iwabuchi, M.; Kuwayama, T.; Muto, T. (Niigata Univ. Sch. Medicine, Niigata, Japan). *Gastroenterol. Jpn.* 12(4):324; 1977.

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M. (Dept. Pediatrics, Univ. California, San Francisco, CA). *Clin. Res.* 26(2):174A; 1978.

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1669 SURGICAL THERAPY OF SEVERE CHRONIC CONSTIPATION. (Ger.) Streuli, H. K.; Fartab, M. (Kantonsspital, CH-5000 Aarau, Switzerland). *Schweiz. Med. Wochenschr.* 107(26):907-912; 1977.

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1671 EVALUATION OF ANTIBACTERIAL THERAPY IN GASTROINTESTINAL FORMS OF SALMONELLOSIS. (Rus.) Tumanov, F. A.; Yushchuk, N. D. (N. A. Semashko Moscow Stomatology Inst., Moscow, USSR). *Sov. Med.* (5):64-67; 1977.

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1673 MOTOR EVACUATORY FUNCTION OF THE INTESTINES IN PATIENTS WITH SALMONELLOSIS. (Rus.) Yushchuk, N. D.; Utekhin, V. A. (Moscow Medical Stomatology Inst., Moscow, USSR). *Sov. Med.* (5):137-138; 1977.

1674 SECONDARY HYPERLIPOPROTEINEMIA. (Ger.) Bruns, W. (Zentralinstitut fur Diabetes, Greifswalder Strasse 11a, DDR-2201 Karlsburg, E. Germany). *Z. Gesamte Inn. Med.* 32(9):133-137; 1977.

1675 SALIVARY CALCINOSIS. (Fre.) Laudenbach, P.; Bonneau, E.; Top, T.; Tran, C. P. (Centre Hospitalier de Bicetre, 78, rue General-Leclerc, F 94270 Le Kremlin-Bicetre, France). *J. Radiol. Electrol. Med. Nucl.* 58(6/7):413-418; 1977.

See also, 1035, 1036, 1040, 1059, 1072, 1073, 1076, 1083, 1090, 1114, 1115, 1116, 1118, 1129, 1139, 1217, 1232, 1244, 1265, 1270, 1271, 1274, 1279, 1298, 1318, 1341, 1390, 1473, 1606, 1617.

GENERAL

Parasitology

1676 HUMAN CHRONIC MANSONIAN SCHISTOSOMIASIS--CELL PROLIFERATION AND FIBRE FORMATION IN THE HEPATIC SINUSOIDAL WALL: A MORPHOMETRIC, LIGHT AND ELECTRON-MICROSCOPY STUDY. (Eng.) Canto, A. L.; Sesso, A.; De Brito, T. (Faculdade de Medicina de Sao Paulo, Sao Paulo Univ., Sao Paulo, Brazil). *J. Pathol.* 123(1):35-44; 1977.

The correlation between sinusoidal cells and reticulin fiber proliferation was studied in human chronic schistosomiasis mansoni. A morphometric, light and electron microscopic study of the sinusoids in biopsy or necropsy specimens from 10 patients without hepatic disease, and 19 patients with schistosomiasis (10 with the intestinal form not exhibiting hypertension, and 9 with the hypertensive, hepatosplenic form) showed a sequence of events beginning with an increased number of cell nuclei, particularly evident at the centrilobular region. This was followed by deposition of reticulin fibers along the perisinusoidal space. Later, increased numbers of cells and reticulin fibers were seen throughout the lobule. Other findings, such as reduplication of the sinusoidal lining and the focal appearance beneath the cell layer of the thin discontinuous membrane, also occurred. This perisinusoidal fibrosis may add an element of heightened intrasinusoidal pressure to the perisinusoidal hypertension already present. The hepatic sinusoids in schistosomiasis mansoni receive mainly arterial blood. High intraluminal pressure may be one explanation of the perisinusoidal fibrosis in these cases. However, as

it possibly occurs in primary sinusoidal portal hypertension, chronic immunological stimulation leading to proliferation of Kupffer cells and lipocytes may be an alternative mechanism. Whatever the pathogenetic mechanism, the livers of patients with chronic schistosomiasis mansoni exhibit a progressive sinusoidal reaction to injury.

1677 CONTROL OF Schistosoma mansoni TRANSMISSION BY CHEMOTHERAPY IN ST. LUCIA. I. RESULTS IN MAN. (Eng.) Cook, J. A.; Jordan, P.; Bartholomew, R. K. (Ministry Health, P.O. Box 93, Castries, St. Lucia, West Indies). *Am. J. Trop. Med. Hyg.* 26(5, Part 1):887-893; 1977.

Two-year results of a program for the control of schistosomiasis in St. Lucia are presented. *Schistosoma mansoni* transmission was controlled by treating all infected persons in Marquis Valley (population about 3,100), St. Lucia with hycanthone; children under 3 yr or weighing less than 15 kg received niridazole or lucanthone hydrochloride. Excluding 26 pregnant patients, 709 of 729 persons who were found to be infected received treatment the first year. Six hundred and seventy-seven patients were given a single injection of hycanthone (2.5 mg/kg), and the same treatment was administered to 150 patients in the second year. Side-effects were not severe; the major side-effect, vomiting, occurred in about 22% on both occasions. In villages with initially high transmission rates, the incidence of new infections in children 0 to 14 yr fell from 20.8% before chemotherapy to 7.4% after 1 yr and to 3.7% after 2 yr. This pattern was significantly different from that in the comparison area where no control scheme exists. Chemotherapy alone appears to be a rapid effective, and comparatively inexpensive method of controlling *S. mansoni* transmission in St. Lucia.

1678 CONTROL OF Schistosomiasis mansoni TRANSMISSION BY CHEMOTHERAPY IN ST. LUCIA. II. BIOLOGICAL RESULTS. (Eng.) Christie, J. D.; Upatham, E. S. (Ministry Health, P.O. Box 93, Castries, St. Lucia, West Indies). *Am. J. Trop. Med. Hyg.* 26 (5, Part 1):894-898; 1977.

Studies were carried out to determine whether a schistosomiasis chemotherapy program had any effect on infection rates in the intermediate snail host in St. Lucia, *Biomphalaria glabrata*. Chemotherapy of all persons infected with *Schistosoma mansoni* was begun in Marquis Valley, St. Lucia, in March 1974. From January 1972 to the start of chemotherapy, the infection rate in field *B. glabrata* collected in the valley was 1.09% (117/10,736), and the rate in sentinel *B. glabrata* was 1.48% (56/3,790). From March 1974 through December 1975, no infections were detected in either field snails (11,742 collected) or sentinel snails (3,230 exposed). The accumulated data suggest that, because of differences in topography and average annual rainfall, *S. mansoni* transmission occurs in this valley during the rainy season, whereas in other St. Lucian valleys under study it occurs during the dry season.

1679 A STUDY OF THE PRESENCE OF CIRCULATING IMMUNE COMPLEXES IN SCHISTOSOMIASIS. (Eng.) Smith, M. D.; Verroust, P. J.; Morel-Maroger, L.; Geniteau, M.; Coulaud, J. P. (Hopital Tenon, 75020 Paris, France). *Trans. R. Soc. Trop. Med. Hyg.* 71(4):343-348; 1977.

The sera of 51 patients with schistosomiasis were studied for the presence of circulating immune complexes (IC) using two methods, inhibition of complement-coated sheep RBC-anti Ig complex (EAC) rosette formation and precipitation of radiolabeled complement component C1q (C1q). The percentage EAC rosette inhibition was significantly greater in the total group of patients compared to the control sera. The material inhibiting EAC rosette formation was precipitable by 4% polyethylene glycol, thus excluding the role of C3 fragments and suggesting that inhibition was due to immune complexes. Using precipitation of radiolabeled C1q, significantly high values compared to control sera were only obtained in those patients before treatment or with an active infection ($p=0.003$). The results suggest that material behaving as IC is present in the sera of patients with schistosomiasis and that measurement of the levels of IC may be important in assessing the state of the disease.

1680 IMMUNE RESPONSES DURING HUMAN SCHISTOSOMIASIS MANSONI. II. OCCURRENCE OF EOSINOPHIL-DEPENDENT CYTOTOXIC ANTIBODIES IN RELATION TO INTENSITY AND DURATION OF INFECTION. (Eng.) Sher, A.; Butterworth, A. E.; Colley, D. G.; Cook, J. A.; Freeman, G. L., Jr.; Jordan, P. (Harvard Medical Sch., 250 Longwood Ave., Boston, MA 02115). *Am. J. Trop. Med. Hyg.* 26(5, Part 1): 909-916; 1977.

Plasma samples from St. Lucians were tested for the presence of antibodies that cooperate *in vitro* with normal human WBC in causing cytotoxic damage to schistosomula of *Schistosoma mansoni*. The *in vitro* antibody activity, which has been previously shown to depend on eosinophil effector cells, was detected in 56% of the individuals with known, current *S. mansoni* infections and in 14% of control subjects from the same endemic area. Quantitatively, eosinophil dependent cytotoxic antibody (EDCA) activity, when expressed as the maximum amount of damage to schistosomula induced at high plasma concentration, correlated significantly with the intensity of *S. mansoni* infection as determined by fecal egg count, the highest levels of activity occurring in patients with stool counts of 60 eggs/ml or greater. In addition, plasma EDCA activity correlated with the *in vitro* blastogenic responsiveness of patients' lymphocytes to three different parasite antigen preparations. In contrast, titrations of EDCA activity failed to reveal a relationship between EDCA titer and the most recent egg count performed on each subject. However, a significant correlation was observed when titers were compared to egg counts averaged over a 3-yr period. Neither maximal EDCA activity nor titer correlated with the duration of known schistosome infection.

1681 IMMUNE RESPONSES DURING HUMAN SCHISTOSOMIASIS MANSONI. III. REGULATORY EFFECT OF PATIENT SERA ON HUMAN LYMPHOCYTE BLASTOGENIC RESPONSES TO SCHISTOSOME ANTIGEN PREPARATIONS. (Eng.) Colley, D. G.; Hieny, S. E.; Bartholomew, R. K.; Cook, J. A. (Veterans Admin. Hosp., Nashville, TN 37203). *Am. J. Trop. Med. Hyg.* 26(5, Part 1):917-925; 1977.

The capability for *in vitro* blastogenesis of lymphocytes from 38 patients with schistosomiasis mansoni was tested against phytohemagglutinin (PHA), *Candida albicans* extract, and soluble preparations of schistosome eggs (SEA), adult worms (SWAP), or cercariae (CAP). When patients' lymphocytes were cultured in medium that contained 5% human (homologous) normal (uninfected) serum, they responded well to PHA and *Candida* extract. The responses induced by SEA were maximal in patients with early *Schistosoma mansoni* infections, while reactivity against SWAP and CAP increased during chronic infection. These responses, induced by the schistosome-derived antigenic preparations, were suppressed if the homologous-normal serum supplement of the culture medium was replaced with either the patient's own (autologous) serum, or that of another patient infected with *S. mansoni*. All sera were heat-inactivated (56 C for 30 min) prior to use. In contrast, responses against the nonspecific mitogen (PHA) and the unrelated antigen (*Candida* extract), were not altered by these changes of the serum supplementation of the media. The degree of suppression by patient serum was not changed by increasing the serum percentage in the medium from 5% to 25%. The suppressive effects of patient sera on responses induced by SEA and SWAP were increased in relation to the duration of the serum donor's *S. mansoni* infection. Preincubation of lymphocytes in suppressive patient sera for 30 min at 37 C did not reduce the expected level of responsiveness if the cells were subsequently cultured in homologous-normal serum supplemented medium. The data indicate that during *S. mansoni* infection, patients develop serum component(s) that specifically interfere with the responsiveness of their lymphocytes in regard to certain schistosome-derived antigenic preparations. The immunoregulatory events described could participate in the modulation of immunopathology, the maintenance of chronic adult worm survival, and the prevention of full expression of protective immune responses.

1682 SINGLE-DOSE TREATMENT OF GIARDIASIS WITH TINIDAZOLE [Letter to Editor]. (Eng.) Danzig, S.; Hatchuel, W. L. F. (Alexandra Health Center and Univ. Clinic, P.O. Bergvlei, Tvl, South Africa). *S. Afr. Med. J.* 52(18):708; 1977.

1683 HEPATIC LINGUATULIASIS IN MAN. (Fre.) Lapierre, J.; Tourte-Schaefer, C.; Vinh Hien, T.; Holler, C.; Bouchard, J.; Deslignieres, S.; *et al.* (Hopital Cochin, 27, rue du Faubourg Saint-Jacques, Paris 14e, France). *Bull. Soc. Pathol. Exot.* 69(5):450-456; 1976.

1684 CLINICAL ASPECTS OF AMEBIASIS. (Ger.) Gyr, K. (Departement fur Innere Medizin, Universitat Basel, Basel, Switzerland). *Schweiz. Med. Wochenschr.* 107(18):639-640; 1977.

1685 EXPERIMENT WITH HYCANTHONE IN 3,100 PATIENTS WITH SCHISTOSOMIASIS MANSONI. (Por.) Buaiz, V.; Sandoval Goncalves, C.; Zanotti, W. M.; Semedo Boni, E.; Pereira, F. E. L. (Dep. de Clin. Med., Univ. Fed. do Espirito Santo, Brazil). *Rev. Assoc. Med. Bras.* 22(5):171-174; 1976.

1686 PERSONAL EXPERIENCE WITH HYCANTHONE AND OXAMINIQUINE IN THE TREATMENT OF SCHISTOSOMIASIS MANSONI. (Por.) Pedro, J. R.; Amato Neto, V. (Departamento de Clinica Medica, Universidade Estadual de Campinas, Campinas, Brazil). *Rev. Bras. Clin. Ter.* 5(8):327-330; 1976.

See also, 0798, 0962, 1052, 1107, 1646, 1650, 1668.

GENERAL

Regional Enteritis

1687 CROHN'S DISEASE INVOLVING THE COLON: AN AUDIT OF CLINICAL MANAGEMENT. (Eng.) Allan, R.; Steinberg, D. M.; Williams, J. A.; Cooke, W. T. (General Hosp., Birmingham B46NH, England). *Gastroenterology* 73(4, Part 1):723-732; 1977.

The clinical management of 204 patients with Crohn's disease involving the colon who have been reviewed at 3- to 6-month intervals for up to 34 yr (mean 13.1 ± 8.7 yr) are described. The results were analyzed to define the morbidity, mortality, and long-term prognosis of the disorder. Despite a

high morbidity, the long-term prognosis was good. Although the mean interval since the onset of symptoms was more than 13 yr, 77% of the patients were symptom-free and 60% had normal laboratory indices. This analysis suggests that until the etiology of Crohn's disease is established and a specific therapy is developed, good results can be obtained with regular medical review and appropriate surgical treatment with the limited use of corticosteroids and the minimal use of immunosuppressive agents or total parenteral nutrition.

1688 SELECTIVE IgA DEFICIENCY AND CROHN'S DISEASE: REPORT OF TWO CASES. (Eng.) Hodgson, H. J. F.; Jewell, D. P. (Royal Free Hosp., London, England). *Gut* 18(8):644-646; 1977.

Two patients with Crohn's disease and selective IgA deficiency are described. Serum IgA was undetectable in each case, and immunoperoxidase studies of the lamina propria showed a gross diminution of IgA-bearing plasma cells. Peripheral blood lymphocytes, however, showed normal numbers of IgA-bearing lymphocytes (2.3% and 6.8% of total lymphocytes in each patient, resp.). The typical clinical course and histology in these two patients suggest that IgA-mediated responses in the mucosa are not involved in the pathogenesis of Crohn's disease.

1689 HLA AND B-CELL ANTIGENS IN PATIENTS WITH CROHN'S DISEASE [Abstract]. (Eng.) Cohen, Z.; Wolfe, E.; Papasteriadis, C.; Werner, C.; Festenstein, H. (London Hosp. Medical Coll., London, England). *Clin. Res.* 25(5):687A; 1977.

1690 STUDIES OF SPECIFIC AND NONSPECIFIC RESISTANCE IN CROHN'S DISEASE [Abstract]. (Eng./Ger.) Heymer, B.; Feyerabend, G.; Haferkamp, O.; Herfarth, C. (Abt. fur Pathologie der Universitat, Steinhovelstr. 9, D-7900 Ulm, W. Germany). *Langenbecks Arch. Chir.* 345:587; 1977.

1691 SURGICAL THERAPY OF CROHN'S DISEASE [Abstract]. (Eng./Ger.) Pridun, N.; Olbert, F.; Kobinia, G.; Russe, O. J. (1. Chir. Abt. des KH der Stadt Wien-Lainz, Wolkersbergenstr. 1, A-1130 Wien, Austria). *Langenbecks Arch. Chir.* 345:587-588; 1977.

1692 PROBLEMS OF ACUTE POSTOPERATIVE EXACERBATION IN CROHN'S DISEASE [Abstract]. (Eng./Ger.) Krieg, H.; Gronninger, J. (Chirurgische Univ.-Klinik, Langenbeckstrasse 1, D-6500 Mainz 1, W. Germany). *Langenbecks Arch. Chir.* 345:588; 1977.

1693 PATIENT'S AND DOCTOR'S DELAYS IN THE DIAGNOSIS OF CROHN'S DISEASE [Abstract]. (Eng.) Jarnerot, G.; Pihl, O.; Aberg, C. (Region Hosp., Linkoping, Sweden). *Scand. J. Gastroenterol.* 12(Suppl. 45):43; 1977.

See also, 1235, 1262, 1290, 1301, 1318, 1320, 1323, 1326, 1328, 1329, 1331.

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